Edward M. Hodder

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A RECORD OF PRACTICAL OBSERVATIONS, AND ANATOMICAL AND CHEMICAL RESEARCHES CONNECTED WITH THE INVESTIGATION AND TREATMENT OF DISEASE.

EDITED BY

LIONEL S. BEALE.

STORAGE

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P.M.—Wt. of heart 16 oz., auricles dilated, left ventricle hypertrophied. Mitral valve very thick, its edges cartilaginous.

Carbonate of ammonia and chloric ether on adm., then with diuretics and elaterium. Brandy and wine up to 8 oz.

Inflammation of knee-joint.—E. B., æt 23. Chandler. Adm. Dec. 15, 1862. Disch. Jan. 21. In H. 9 days. *Recovery.* Dr. Beale, vol. 5, p. 88, Strumous. Not very temperate. Pain, tenderness, and swelling of right knee, following a long walk. P. 116. No Pain after 16th day.

Chiefly, iodide of potassium, colchicum, and bicarb. of potass, and afterwards with ammonio-citrate of iron, and cod-liver oil.

Subacute Rheumatism.—T. H., æt. 35. Hospital Porter. Adm. Feb. 19, Disch. Feb. 28. In H. 9 days. *Recovery.* Dr. Garrod, not in case book. Very mild attack. No heart affection.

Iodide of potassium, and bicarbonate of potass.

Subacute Rheumatism.—L. E., æt. 28. Cook. Adm. Feb. 18. Disch. Mar. 6. In H. 16 days. *Recovery.* Dr. Beale, vol. 6, p. 96. Acute Rheumatism 6 yrs. ago (5 wks.) Prev. ill. 3 days. Feet and ankles painful and swollen. Moderate sweating. Basic systolic bruit over heart on adm.

Bicarbonate of potass, quinine and opium.

Subacute Rheumatism.—S. G , æt. 20. Servant. Adm. Feb. 19. Disch. Mar. 4. In H. 13 days. *Recovery.* Dr. Beale, vol. 5, p. 129. First attack. Prev. ill. 4 days. Left wrist and right shoulder affected. No Cardiac disease.

Liquor ammoniæ acetatis and chloric ether, and purgatives.

Subacute Rheumatism & Chronic Bronchitis.—A. D., æt. 68. Orange-woman. Admitted Dec. 10, 1862. Disch. Feb. 7. In H. 59 days. *Relieved.* Dr. Beale, vol. 6, p. 45. Acute rheumatism 21 yrs. ago. Since then has lived badly, and been much exposed to wet and cold. Cough 6 months. Prev. ill. 1 month, with pain and swelling of right ankle. On adm. œdema of legs and feet. P. 76. Left shoulder afterwards affected. No cardiac disease.

Liquor ammoniæ acetatis, chloric ether, bicarbonate of potass, and decoction of senega. Afterwards with muriated tincture of iron. Blisters to left shoulder.

Subacute Rheumatism.—J. G., æt. 27. Gardener's Labourer. Adm. Feb. 13. Disch. Feb. 25. In H. 12 days. *Recovery.* Dr. Garrod, vol. i, p. 50. Two former attacks 9 yrs. and 18 mon. ago. Shortness of breath and cough 18 mon. Prev. ill. 14 days. No exposure to wet. Commenced in left aukle; wrists, elbows, shoulders and knees attacked about 11th day. Diastolic bruit over base of heart. Slight sweating. Excess of urea in urine. P. 82.

Quinine and bicarbonate of potass.

Subacute Rheumatism —W. L., æt. 30. Labourer. Adm. Feb. 16. Disch. March 11. In H. 23 days. *Recovery.* Dr. Beale, vol. 6, p. 84. First attack 11 yrs. and second 4 yrs. ago. Prev. ill 7 days. Ankles and knees first attacked, and then wrists. Systolic bruit over apex of heart on 15th day.

Bicarbonate of potass, quinine, and opium.

Subacute Rheumatism.—E. O. Cook, æt. 19. Adm. Feb. 11. Disch. Feb. 25. In H. 14 days. *Recovery.* Dr. Garrod, vol. 2, p. 38. Hard worked. Scarlet fever 5 years ago. followed by acute rheumatism ; no carditis Prev. ill. 11 days, with præcordial pain and dyspnœa. On 4th day pain and swelling of left ankle, and acid sweats, followed by pain in knees and hips. P. 84. Systolic bruit over base of heart; venous murmur in neck. Convalescent on 21st day.

Bicarbonate of potass (3ss doses), and quinine for half the time, and then with quinine, sulphate of iron, and sulphuric acid.

Subacute Rheumatism.—W. J., æt. 21. Pugilist. Adm. Feb. 14. Disch. Mar. 4. In H. 18 days. *Recovery.* Dr. Johnson, vol. 15, p. 97. A hard drinker.

Pains in limbs 4 mon. after sleeping on grass for 5 nights. Prev. ill. 18 days. Knees, ankles, hips, shoulders, elbows, wrists, affected in succession. Cough and profuse expectoration. Pain and increased dulness over heart. Basic systolic bruit.

Bicarbonate of potass, quinine, and opium, and afterwards with carbonate of ammonia, and ammonio-citrate of iron.

Subacute Rheumatism.—Cath. P., æt. 46. Married. Adm. Nov. 25, 1862. Disch. Jan. 17. In H. 53 days. *Recovery.* Dr. Budd, vol. 73, p. 100. No family history of rheumatism or gout. Drinks spirits. Slight attack of rheum. 3 yrs. ago. Prev. ill. 2 days. Knees and ankles first affected, afterwards wrists and shoulders. A little pain in left shoulder on Jan. 9. Moderate sweating. No cardiac disease. Highest pulse 130.

Bicarbonate and nitrate of potass, and colchicum and opium. Afterwards with bicarbonate of potass and quinine.

Acute Rheumatism & Bronchitis.—A. B., æt. 32. Needlewoman. Adm. Dec. 22, 1862. Disch. Jan. 31. In H. 40 days. *Recovery.* Dr. Johnson, vol. 16, p. 46. Acute rheumatism 15 yrs. ago. Shortness of breath 5 yrs. Bronchitis and dropsy of feet and ankles 3 yrs. ago. On adm. joints of right side inflamed. Systolic bruit over apex. On 18th day after adm. double bruit over base. Moist sounds over lungs. Highest pulse 104: highest resp. 22.

Bicarbonate and nitrate of potass, chloric ether and ammonio-citrate of iron. Turpentine stupes to chest.

*Acute Rheumatism.—S. A. de K., æt. 12. Adm. Jan. 22. Disch. Mar. 21. In H. 58 days. *Recovery.* Dr. Johnson, vol. 15, p. 75. Very anæmic. No exposure to cold. Prev. ill. 5 days. Commenced in left ankle, then in left knee, right ankle, right knee, hips, shoulders, elbows and wrists. P. 100. Cardiac dulness slightly increased. Basic systolic bruit. On 25th day double bruit at base. Pulse collapsing. Convalescent on 40th day.

Bicarbonate of potass, quinine and opium. Quinine and sulphate of iron during convalescence. Blister to right shoulder.

*Acute Rheumatism.—E. G., æt. 22. Servant. Adm. Dec. 16, 1862. Disch. Jan. 31. In H. 60 days. *Recovery.* Dr. Johnson, vol. 16, p. 27. Cough and occasional hæmoptysis, 3 yrs. Catamenia irregular. Prev. ill. 14 days, after getting wet through on two successive days. Ankles, knees, elbows, and shoulders affected. Systolic bruit loudest over base. Profuse sweating. Convalescent on 30th day. Highest pulse 124: highest resp. 48.

Bicarbonate of potash, and quinine, and opium, and ammonio-citrate of iron.

Acute Rheumatism.—John M., æt. 13. No occ. Adm. Feb. 5. Disch. Feb. 18. In H. 13 days. *Recovery.* Dr. Garrod, vol. 1, p. 42. Strumous disease of hip, 6 yrs. Prev. ill. 7 days. Pain and swelling of left knee and right wrist, afterwards of right elbow. Pain over heart till 8th day. Systolic bruit over apex on adm. Profuse sweating till 14th day. Highest pulse 112.

Bicarbonate of potass and quinine.

*Acute Rheumatism.—H. B., æt. 17. Nursemaid. Adm. Jan. 15. Disch. Mar. 14. In H. 59 days. *Recovery.* Dr. Garrod, vol. 2, p. 4. Prev. health good. First attack. Prev. ill. 14 days. Redness, pain, and swelling of left shoulder, after getting wet; then of wrists, hips, knees, and ankles. Profuse acid sweats. On adm. knees and right ankle inflamed. Knees well on 19th day, ankle on 54th day. No cardiac disease.

Alkalies and quinine for 16 days. Then iodide of potassium for 24 days, and syrup of iodide of iron during convalescence.

*Acute Rheumatism.—G. F., æt. 55. Coach-driver. Adm. Jan. 31. Disch. Mar. 14. In H. 42 days. *Recovery.* Dr. Garrod, vol. 1, p. 37. Intemperate. Thirty years ago general dropsy, after exposure to cold. Prev. ill. 3 wks. Began in ball of left great toe, then ankles, knees, shoulders, elbows, wrists, and small

joints of hand. Profuse sweating. P. 108. No family history of gout. No excess of uric acid in blood serum. No pain remaining on 55th day.

Bicarbonate of potass (℈ii) *and quinine. Then with carbonate of ammonia* (10 *gr. doses*), *and quinine, and decoction of bark. Brandy* 6 *oz. afterwards changed for gin.*

Rheumatism.—W. L., æt. 33. Compositor. Adm. Dec. 2, 1862. Disch. Jan. 26. In H. 55 days. *Much relieved.* Dr. Budd, vol. 74, p. 119. Inflammation of ball of right great toe during last 3 winters. Gonorrhœa 1 month. Prev. ill. 3 wks. with inflammation of ball of right great toe 37 days, ankles (76 days), and small joints of hand (20 days). Heart unaffected. Still some pain in ankles on discharge.

Principally, bicarbonate and iodide of potassium, and decoction of bark. Afterwards with guiacum, large doses of iodide of potass, and quinine, and acid.

*Sciatica.—J. C., æt. 48. Farrier. Adm. Dec. 12, 1862. Disch. Jan. 28. In H. 48 days. *Recovery.* Dr. Budd, vol. 73, p. 116. Exposed to great variations of temperature. Prev. ill. 7 weeks. Pain in hip, and back of right thigh, and calf, increased by pressure (88 days).

Quinine (*gr* i *doses*), *and subdermic injection of morphia, at first Then with* 5 *gr. doses of quinine gradual y increased to* 15 *grs. t. d. This relieved the pain, but caused slight cinchonism after two days, and was then left off. The pain returning, he was put on quinidine,* 15 *grs. t. d. This did not cause cinchonism and he recovered perfectly under it.*

*Sciatica.—J. W., æt. 52. Carpenter. Adm. Dec. 12, 1862. Disch. Jan. 14. In H. 33 days. *Recovery.* Dr. Johnson, vol. 15, p. 33. Prev. ill. 1 month. Pain along left sciatic nerve, extending down leg. Commenced suddenly. Duration of pain 61 days. Bronchitis on adm.

Subdermic injection of morphia, and blisters to hip. Carbonate of ammonia, and chloric ether at first, and then hydrochlorate of ammonia, and tincture of sesquichloride of iron.

Variola.*—M. P., æt. 38. Hospital-nurse. Adm. Feb. 24. Disch. Mar. 19. In H. 23 days. *Recovery.* Dr. Beale, vol. 6, p. 106. Attended a fatal case of small-pox a fortnight bef. adm. Prev. ill. 6 days, with frontal headache, and pain in stomach and back. Shivering on Feb. 21. Eruption on Feb. 23; slight, not confluent, commencing on right shoulder, then on face and body.

Ammonia and chloric ether. Nitric acid and bark during convalescence.

Chorea.—Ellen W., æt. 8½. Adm. Jan. 5. Disch. Jan. 6, at request of mother. Dr. Garrod, not in case-book. Chorea 3 wks. after a fright, commencing in legs. No heart-disease.

Not treated.

*Chorea and Acute Rheumatism.—L. F., æt. 17. Domestic servant. Adm. Sept. 17, 1862. Disch. Jan. 23. In H. 128 days. *Recovery.* Dr. Budd, vol. 73, p. 30. Catamenia suppressed 2 mon. Prev. ill. 3 wks. A fright, followed by violent choreic movements till 48th day; then slight twitching of hands and face till Jan. 14. Acute rheumatism on 9th day after adm. (37 days) commencing in left wrist, then in ankles and shoulders. Profuse sweating. Systolic bruit over apex of heart.

Quinine, sesquichloride of iron, and shower-baths. During the rheum. alkalies and bark, morphia, and blisters above joints. After Jan. 1, *sulphate of iron* (8 *grs. t. d. gradually increased to* 16 *grs. t. d.*).

* The patient could not be received at the Small Pox Hospital. See note on page 91.

Mental Derangement.—J. B., æt. 22. Coach-painter. Adm. Dec. 31, 1862. Disch. Jan. 16. In H. 16 days. *Not relieved.* Dr. Johnson, vol. 15, p. 43. Very intemperate. Not violent. Various delusions.
Quinine, and sesquichloride of iron and opium.

Mental Derangement, Epilepsy.—M. W., æt. 33. Groom. Adm. Jan. 19. Disch. Feb. 5. In H. 17 days. *Not relieved.* Dr. Johnson, vol. 16, p. 79. Had syphilis. Prev. ill. 1 yr. Epileptic fits gradually increasing in frequency. Pain in forehead, impaired memory, incoherence, violent at night. No fit while in K. C. H.
Opiates, iodide of potassium and quinine.

Cephalalgia.—E. H., æt. 33. Labourer. Adm. Jan. 27. Disch. Feb. 4. In H. 8 days. *Not relieved.* Dr. Johnson, vol. 16, p. 93. Syphilis 11 yrs. ago. Headache, vertigo, and failure of sight of left eye (2 yrs.). Pupils equal.
Iodide of potassium and morphia.

*Disease of Larynx.—E. C., æt. 40. Employed in a seed warehouse. Adm. Mar. 13. *Died* on Mar. 14. Dr. Johnson, vol. 15, p. 102. Cough during past winter. Prev. ill. 1 wk. with increase of cough and loss of voice. On adm. breathing difficult, not stridulous—no pain over larynx—aphonia. P. above 100. Chest resonant. An attack of spasm a few hours after adm. Tracheotomy. Death shortly after insertion of tube. P.M.—Posterior part of right true vocal cord pushed up by a small hard tumour just below it, size of a large pea, with ulcerated surface, and scirrhous appearance on section. Laryngeal mucous membrane natural. Lungs congested.
Brandy, liq. morph. mur. mxx., steam inhalation, and operation as above.

Mental Derangement.—A. C., æt. 17. Servant. Adm. Jan. 27. Disch. Mar. 28. In H. 60 days. *Improved.* Dr. Johnson, vol. 15, p. 59. Catamenia irregular. Leucorrhœa. Religious delusion. Operated on for strabismus of right eye on Mar. 10.
Opiates, valerian, quinine and sesquichloride of iron, and asafœtida.

Headache.—J. G., æt. 40. Fly Driver. Adm. Jan. 14. Disch. Jan. 28. In H. 14 days. *Relieved.* Dr. Johnson, vol 15, p. 57. Prev. ill. 6 mos., with nausea, loss of appetite, and pain in back of head. Præcordial pain, 4 mos. Hæmatemesis 19 days bef. adm. Muscular tremors, vertigo and sleeplessness.
Opiates and quinine.

*Arachnitis.—T. S., æt. 16. Porter. Adm. Mar. 9. *Died* on Mar. 9. Dr. Johnson, vol. 15, p. 88. Excitable. On Mar. 8 headache and nausea, but able to work a little till afternoon of next day, when the headache increased, he became very restless, and shortly afterwards delirious. On adm. (about 7·30 p.m.) pupils dilated; severe headache; great restlessness; replies "Yes, sir," to almost every question; delirium. Sudden death about ½ an hour after adm., preceded by a slight convulsive attack. P.M.—Pus beneath dura mater, and on surface of convolutions. Kidney congested.

*Delirium Tremens.—M. Q., æt. 39. Betting Man. Adm. Mar. 1. Disch. Mar. 11. In H. 10 days. *Recovery.* Dr. Garrod, vol. 1, p. 60. Sometimes drinks Oij brandy in a day. Drunk 5 days. Prev. ill. 1 day. Nausea; violent tremors; spectral delusions; sleepless. P 120. Noisy on 2nd day after adm. Slept after. Tr. Digit. ℥ij.
A purge; carbonate of ammonia; Liq. Morph. Mur. ℥ij *(in 2 doses); opii gr.* i. *3tiis horis (4 doses). Tr. Digit.* ℥ij, *repeated next night. Porter* ℥i, *brandy* ℥ij.

*Coma (Epileptiform)—Pleurisy.—M. B., æt. 22. Prostitute. Adm. Jan. 8. Disch. Mar. 4. In H. 55 days. *Recovery.* Dr. Garrod, vol. 1, p. 13. Had one child. Occasional headaches (2 yrs.). Catamenia absent (5 wks.). Much mental anxiety of late. Between 2 and 3 p.m. on Jan. 7, while talking to a friend fell down in convulsions, which lasted all night and were followed by stupor. On adm. (2nd day) eyes closed; complete unconsciousness; pupils equal, and sensible to light; slight trismus. No paralysis of limbs. Swallows liquids. Respiration quiet. P. 108. Basic systolic bruit over heart. Urine retained. No albumen. Twenty-three convulsive attacks the next day, between 6 a.m. and 1 p.m. Pupils sensible to light; mucous rattle in throat; motions passed under her; incontinence of urine (11 days). Return of convulsions at 6 p.m.; they ceased at 3 p.m. the next day (4th), and she then, for the first time since admission, noticed what went on around her. On 5th day she answered questions, but was delirious for 5 days following. On 7th day, p. 120; dulness over right base; and on 8th day friction sound over right base (5 days). Violent delirium on 12th day. Rapid improvement after 14th day, though great anæmia remained. Reappearance of catamenia on Feb. 23.

A turpentine enema on adm., and then nothing else, except brandy and beef tea, till 4th day, when 2 drops of croton oil with castor oil, an enema with 15 grs. quinine, and an enema with ℥ss of Tr. asafœtida were exhibited. She improved after this, and the asafœtida was repeated on the three following days. Then carbonate of ammonia and bark, and during convalescence quinine and sulphate of iron, and fer reduit. Brandy 6 oz.; increased to 12 oz. on 6th day, and reduced to 6 oz. on 22nd day.

*Paraplegia.—G. W., æt. 29. Soldier. Adm. Dec. 26, 1862. *Died* on Mar. 11. In H. 75 days. Dr. Johnson, vol. 16, p. 59. Primary syphilis 8 yrs. ago; no secondary symptoms. Vertigo at Gibraltar 16 mos. ago: 1 mo. after this, sudden loss of motion and sensation in right arm and leg; face affected; partial recovery. Five months ago, sudden motor palsy of right side; speech slightly affected. Two months ago fell down; no loss of consciousness; legs palsied; sensation and speech unaffected. Tremors of right leg, and slight rigidity of right biceps femoris. Motions and urine passed under him for 26 days after adm. Then rapid improvement, and on 34th day after adm. could walk with a stick. Death sudden, preceded by headache (1 wk.). P. M.—Recent hœmorrhage at base of brain, extending down cord; softening of lower dorsal region of cord. Oily granular cells through whole length of cord.

Quinine and sesquichloride of iron for 5 days; then iodide of potassium, 61 days. During the last wk. quinine and sulphuric acid.

Constipation. Epilepsy.—Clara S., æt. 7. Adm. Jan. 5. Disch. Jan. 18. In H. 13 days. *Relieved.* Dr. Johnson, vol. 16, p. 71. Always epileptic. Fits have diminished in number and severity the last year or two. Drags right leg slightly in walking. Constipation and vomiting.

Castor oil.

*Hemiplegia.—C. R., æt. 43. Butcher's wife. Adm. Jan. 19. Disch. March 11. In H. 51 days. *Slightly Relieved.* Dr. Johnson, vol. 16, p. 81. Scarlet fever when a child. Previous illness 14 days. Mental anxiety. Fell down while at work. Loss of consciousness. Right side paralysed. Complete loss of sensation in right half of body. Mouth and tongue a good deal affected. Slight rigidity of biceps of arm. Basic systolic bruit over heart. Trace of albumen in urine. Gout on 7th day after adm. Slight improvement of leg.

Treatment various, chiefly iodide of potassium, and quinine and iron. Blisters to nape of neck.

*Hemiplegia.—W. F. M., æt. 38. Law clerk. Adm. Dec. 19. Disch. Jan. 17. In H. 29 days. *Relieved.* Dr. Johnson, vol. 16, p. 36. A sun-stroke while in China 10 years ago. Giddiness, dimness of sight, and pains in head, 2 months.

Mental anxiety. During dinner on Dec. 19, sudden loss of consciousness. On adm. unconscious (3 days), loss of motion, and sensation on left side. Not much face palsy. Reflex action unimpaired. No rigidity. Heart healthy. Sensation began to return in arm on 5th day. Recovery nearly complete.

Sal volatile and a purge. Then chloric ether and muriatic acid, and afterwards sesquichloride of iron.

*Paralysis.—T. W., æt. 48. Saddler. Adm. Jan. 21. Disch. Jan. 31. In H. 10 days. *Relieved.* Dr. Beale, vol 5, p. 103. Formerly a soldier. Intemperate. Prev. illness 14 days. Pains in legs. Thickness of speech. Double vision (for 5 days after adm.). Slight loss of power in left arm. Since readmitted with left hemiplegia.

Dilute nitric acid and chloric ether.

*Hemiplegia.—W. L., æt. 23. Sailor. Adm. Feb. 21. Disch., at his own request, Mar. 9. In H. 16 days. *Relieved.* Dr. Johnson, vol. 16, p. 123. Had cholera 9 years ago. Ague 6 years ago. Syphilis 5 years ago. No secondary symptoms. Prev. illness 7 weeks. Four epileptic fits in 2 days, followed by complete loss of motion and partial loss of sensation in left arm and leg On adm. slight motor palsy of face, tongue, arm, and leg. Since readmitted with *right* hemiplegia.

Iodide of potassium.

*Hemiplegia.—H. C., æt. 50, jeweller. Adm. Oct. 2, 1862. Disch. Feb. 21. In H. 142 days. *Relieved.* Dr. Johnson, vol. 13, p. 241. Intemperate. Subject to gout 16 years. Last attack 5 months ago. Pain in back of head 14 days. On morning of adm., after working all night, giddiness, pain in left temple and sudden left hemiplegia, without loss of consciousness. On adm. in complete palsy of motion; reflex action and sensation perfect; slight rigidity of left biceps of arm; headache; right pupil dilated (6 days); urine albuminous; heart normal; radial arteries rigid. From Oct. 4 to Oct. 9, delirium, torpor, and evacuations passed under him. On Oct. 10, an attack of gout commencing in left foot (47 days). Another attack of gout in feet on December 26, and on Jan. 10 in left knee. Recovery nearly complete.

Quinine and blister to nape of neck, and then ammonio-citrate of iron, chloric ether and colchicum. Blisters to left knee. Brandy 3 *oz.*

*Hemiplegia.—Emma R., æt. 11. Adm. Dec. 14, 1862. Disch. Jan. 31. *Relieved.* In H. 48 days. Dr. Johnson, vol. 15, p. 31. A fright 4 mos. ago, was followed immediately by tremors, and in 3 days by complete loss of power in left arm and leg (9 weeks). On Dec 14 sudden insensibility and left hemiplegia after a meal of roast pork. Face drawn. Sensation unaffected. Slight rigidity of limbs. Systolic bruit over apex of heart. Partial recovery. A second slighter attack of left hemiplegia, without loss of consciousness, on 16th day after adm. Recovery nearly complete.

Grey powder and rhubarb, and sesquichloride of iron.

*Hemiplegia.—Margaret R., æt. 56. Adm. Jan. 16. Disch. March 18. In H. 62 days. *Slightly relieved.* Dr. Beale, vol. 5, p. 96. Typhus 42 yrs. ago. Right hemiplegia 2 years ago; complete recovery in 1 yr. Three mos. ago tingling and loss of power in left leg (1 wk.). Previous illness 5 wks., sudden loss of power in arm and weakness of leg. On day bef. adm. fell down. Slight loss of consciousness. Complete left hemiplegia, with partial loss of sensation. No rigidity. Reflex action unimpaired. Heart normal.

Treated with quinine and sesquichloride of iron; iodide of potassium; galvanism; and occasional purgatives.

W. K., æt. 50. No occupation. Adm. Mar. 29. Discharged Mar. 30. *Recovery.* Dr. Johnson, not in case book. Slight hemiplegia after drinking.

Neuralgia.—G. U., æt. 63. Printer's Labourer. Adm. Feb. 7. Disch. Feb. 25. In. H. 18 days. *Relieved.* Dr. Johnson, vol. 16, p. 111. Intemperate. Much exposed to cold. Acute rheum. 30 yrs. ago. Neuralgia of face (right side) 3 yrs. Inflammation of face (8 days) following application of a horseradish poultice two days bef. adm.

Carbonate of ammonia, chloric ether and bark; then quinine and sesquichloride of iron. Morphia at night, and local application of belladonna.

Neuralgia.—A. R., æt. 37. Servant. Adm. Mar. 4. Disch. Mar. 25. In H. 21 days. *Recovery.* Dr. Beale, vol. 5, p. 136. Neuralgia of left side of face 3 yrs. In K. C. H. in May, 1862, under Dr. Budd, for same complaint.

Sesquichloride of iron and purgatives.

Paralysis of Third Nerve.—W. F., æt. 31. Plasterer. Adm. Jan. 19. Disch. Jan. 20, at his own request. Dr. Johnson. Not in case-book.

Ulceration of Soft Palate.—Ann C., æt. 36. Servant. Adm. Mar. 3. Disch. Mar. 28. In H. 25 days. *Recovery.* Dr. Johnson, vol 15, p. 115. Since her marriage, 16 yrs. ago, loss of health, and pains in bones; no eruption. Sore throat 7 yrs., worse during last 6 mos. On adm. uvula and part of soft palate gone. Ulceration extending to hard palate. Slougning of back of pharynx. Dysphagia.

Iodide of potassium and bark, and local application of nitric acid.

Ulceration of Throat.—S. B., æt. 31. Labourer. Adm. Feb. 28. Disch. Mar. 21. In H. 21 days. *Nearly well.* Dr. Johnson, vol. 15, p. 113. Syphilis 9 yrs. ago, and again 3 yrs. ago. In St. George's 9 mos. ago for secondary eruption. Prev. ill. 3 mos. Ulceration of uvula and right tonsil.

Iodide of potassium, and local application of nitrate of silver, and nitric acid.

Cynanche Tonsillaris.—E. H., æt. 23. Servant. Adm. Feb. 18. Disch. Mar. 11. In H. 21 days. *Relieved.* Dr. Beale, vol. 5, p. 126. Sore throat (14 days) 6 mos. ago. Prev. ill. 9 days. Followed by chronic enlargement.

Chlorate of potass, and sesquichloride of iron. Grey powder and rhubarb twice a week, and sol. A.N. to tonsils.

Cynanche Tonsillaris.—E. S., æt. 17. Servant. Adm. Jan. 22. Disch. Feb. 11. In H. 20 days. *Recovery.* Dr. Beale, vol. 5, p. 108. Prev. ill. 14 days.

Acetate of ammonia and chloric ether, and afterwards dilute nitric acid. Calomel purge. Wine 4 oz.

Colic.—M. S., æt. 26. Commissionaire. Adm. Mar. 5. Disch. Mar. 9. *Recovery.* Dr. Johnson, vol. 15, p. 123. Colic after a long walk.

A warm water enema, opium and purgatives.

*Lead Colic.—Ann F., æt. 32. In white lead factory. Adm. Dec. 29, 1862. Disch. Jan. 21. In H. 23 days. *Recovery.* Dr. Johnson, vol. 16, p. 52. Works in a part of the factory where there is much white-lead dust floating about in the air. Loss of appetite for 3 wks. before adm. Pains in limbs, constipation and gradually increasing abdominal uneasiness and pain for 2 wks. before adm. Marked blue line on gums. No dropping of wrists.

Iodide of potassium; colocynth pills; and colocynth and ext. of nux vomica.

Dysenteric Diarrhœa.—W. J., æt. 40. Bricklayer. Adm. Feb. 19. Disch. Mar. 16. In H. 25 days. *Recovery.* Dr. Beale, vol. 5, p. 127. Diarrhœa (3

wks.) 4 mos. ago. Prev. ill. 8 days. Loose motions containing blood; purged every ½ of an hour at first, and on adm. 3 or 4 times a day. Stools natural on Mar. 6.

Sulphate of copper and opium for 10 days. Then sesquichloride of iron, and castor oil.

*Acute Renal Dropsy.—Caroline C., æt. 36. Adm. Feb. 26. *Died* on Mar. 28. In H. 30 days. Dr. Johnson, vol. 16, p. 127. Dropsy of feet and ankles 10 yrs. ago (4 wks.). Prev. ill. 3 wks. Dropsy of legs. Urine scanty; turbid with lithates; ⅔ths albumen. No thoracic disease. Improved up to 15th day after adm. Then sloughing about left ankle, and back of leg, accompanied by rapid wasting. Death from exhaustion. P. M.—Heart small. Lungs gorged. Liver fatty; wt. 4½ lbs. Kidneys.

Acetate of ammonia and citrate of iron, diuretics, and purgatives. Hot air baths and dry cupping on alternate nights. Right leg punctured on 22nd day after a slough had formed on the left leg.

*Acute Renal Dropsy.—H. A., æt. 27. Carman. Adm. Jan. 31. Disch. Mar. 2. In H. 30 days. *Recovery.* Dr. Garrod, vol. 2, p. 25. Temperate. Much exposed to wet and cold. Prev. ill. 11 days. Pain in belly and right loin (13 days). Vomiting. Scanty, smoky urine; ½ albumen on adm. Dropsy of legs, 24 days.

Cream of tartar and jalap, and mustard poultices to loins. Afterwards sulphate of iron.

*Renal Dropsy.—T. K., æt. 43. Compositor. Adm. Jan. 31. Disch. Mar. 28. In H. 56 days. *Relieved.* Dr. Johnson, vol. 16, p. 95. Syphilis 12 yrs. ago. Prev. ill. 2 mos. Dropsy of feet and ankles spreading upwards to knees. Urine ⅛th albumen, containing blood.

Acetate of ammonia; compound jalap powder; hot air baths; and iron.

*Renal Dropsy.—J. R., æt. 28. Labourer. Adm. Nov. 8, 1862. *Died* on Feb. 26. In H. 110 days. Dr. Johnson, vol. 15, p. 3. Intemperate. Acute rheum. 4 yrs. ago. Prev. ill. 6 wks. Dropsy of face and legs 3 wks. after getting wet. Dyspnœa 1 wk. bef. adm. Anasarca and ascites. Cough and expect. Rhoncus over lungs; dulness over left lower lobe. Heart normal. Urine ½ albumen containing granular and waxy casts, epithelium and blood. A week after adm. 3 epileptic seizures, chiefly affecting left side. Legs punctured on Jan. 8. On Feb. 5 large waxy and granular casts and much oil. Feb. 25, nausea; tongue dry and brown; 10 oz. water in 24 hours. Conscious till a few minutes before death. P. M.—Lungs everywhere adherent. Left ventricle hypertrophied; nitral valve thickened. Much fluid in peritoneum. Commencing atrophy of kidneys.

Dry cupping over loins; hot air baths; hydragogue purgatives, iron, and stimulants. Gin 6 oz.

*Renal Dropsy.—E. J., æt. 29. Tanner. Adm. Dec. 3, 1862. Disch. Jan. 17. In H. 45 days. *Improved.* Dr. Budd, vol. 73, p. 108. Temperate. Previous health good. Prev. ill. 6 wks. Shivering and retching followed by a rash (child with scarlatina in same house). Anasarca; scanty and bloody urine; pain in loins (5 wks.); urine containing oily casts and blood; albumen ½.

Jalapine and colocynth, acetate and carbonate of ammonia, and hot air baths, for a month. Then large doses of jalap and cream of tartar, and sulphate of iron.

Renal Disease.—E B., æt. 43. Groom. Adm. Mar. 11. Disch. Mar. 28. In H. 17 days. *Relieved.* Dr. Johnson, vol. 16, p. 143. Formerly intemperate. Syphilis 15 years ago. In K.C.H. 6 times under Dr. Budd with same symptoms as at present. Frequent vomiting, 10 wks. Cough and slight hæmoptysis. Tenderness on pressure over abdomen. Granular casts in urine; no albumen.

Acetate of ammonia and chloric ether. Afterwards citrate of iron.

*Renal Dropsy.—Lydia M., æt. 16. Adm. Nov. 26, 1862. *Died* on Jan. 11. In H. 46 days. Dr. Johnson, vol. 15, p. 19. Typhus 6 yrs. ago. Prev. ill. 9 mos. Pain in loins, dropsy of legs, and cough. Heart normal. Urine ⅓rd albumen, containing oily casts, and epithelium. Ascites by 19th day after adm. Erysipelas of right thigh on 29th day (5 days). Pain and tenderness over abdomen on 42nd day, followed by sinking. P. M.—A little lymph on peritoneum; fluid in pleuræ and abdomen; scattered tubercles in lungs; fatty degeneration of kidney.

Jalapine and colocynth, hot air baths, iron and stimulants.

*Renal Disease—Gout.—A. M., æt. 53. Needlewoman. Adm. Jan. 2. Disch. Mar. 4. In H. 61 days. *Recovery.* Dr. Garrod, vol. 1, p. 1. Intemperate. Mania 20 yrs. ago (3 yrs.). Two months ago, pain in loins, debility, vomiting. One month ago, debility and giddiness. Pain, œdema, and redness of feet till 6th day after adm. Urine albuminous (4 days); waxy and granular casts. On Feb. 7, excess of uric acid in blood serum. Scabies.

Treatment, various.

Jaundice.—J. S., æt. 9. Adm. Dec. 29, 1862. Disch. Jan. 14. In H. 16 days. *Recovery.* Dr. Johnson, vol. 15, p. 41. Never had rheumatism. Prev. ill. 8 days. Motions pale. No hepatic pain or enlargement. Systolic bruit at apex of heart. Duration of jaundice 18 days.

Sulphate of magnesia and muriatic acid for a week, and then with ammonio citrate of iron.

Jaundice.—W. K., æt. 19. Printer. Adm. Mar. 5. Disch. Mar. 21. In H. 16 days. *Recovery.* Dr. Beale, vol. 6, p. 113. Prev. ill. 1 wk. Slight jaundice and ulcerated sore throat. Motions pale. No hepatic pain, tenderness, or enlargement.

Nitro-muriatic acid, mist, alba, and quinine.

Jaundice.—A. J., æt. 16½. Housemaid. Adm. Dec. 9, 1862. Disch. Jan. 10. *Recovery.* Dr. Budd, vol. 73, p. 110. Hysterical. Pain in right side and occasional constipation 9 mos. Prev. ill. 3 mos. Vomiting, loss of appetite, and pain and tenderness over right hypochondrium. Jaundice on day after adm. (10 days). Liver enlarged. Constipation.

Sulphate and carbonate of magnesia and sal-volatile. Afterwards quinine and sesquichloride of iron.

Jaundice.—W. L., æt. 16. Printer. Adm. Feb. 4. Disch. Feb. 17. In H. 13 days. *Recovery.* Dr. Johnson, vol. 15, p. 87. Works in an atmosphere loaded with steam. Jaundice 1 wk. bef. adm., preceded by nausea and vomiting. Headache. No pain, tenderness, or enlargement of liver. Motions clay coloured. Duration of jaundice, 19 days.

Pil-panis for 1 wk. Then mist-alba and quinine and sulphuric acid.

Jaundice.—H. M'Q., æt. 27. Stationer. Adm. Feb. 23. Disch. Mar. 14. In H. 19 days. *Recovery.* Dr. Johnson, vol. 15, page 109. Acute rheumatism 4 yrs. ago. Nausea and vomiting on Feb. 11. Constipation. Jaundice 1 wk. before adm. Languor. Motions clay coloured. No hepatic pain, tenderness, or enlargement. Systolic bruit over apex of heart.

Sulphate of magnesia; dilute nitro-muriatic acid; and podophyllin and colocynth.

Jaundice.—A. S., æt. 16. Lithographic Printer. Adm. Jan. 20. Disch. Feb. 11. In H. 12 days. *Recovery.* Dr. Garrod, vol. 2, p. 22. Sour risings 1½ yrs. Costive bowels, 3 wks. Prev. ill. 6 days. "Caught cold;" feeling of constriction round belly, and jaundice. Slight hepatic tenderness on day after adm.; no enlargement. Motions pale till 4th day after adm.

Calomel purge, and sulphate of magnesia, and sal-volatile.

*Cirrhosis of Liver.—Eliz. F., æt. 51. Cook. Adm. Feb. 18. *Died,*

Mar. 14. In H. 24 days. Dr. Johnson, vol. 16, p. 119. Been a spirit drinker. No rheum. Prev. ill. 7 wks. Gradual painless enlargement of abdomen. Skin dry; superficial abdominal veins enlarged; considerable ascites, liver pushed up. Systolic bruit over apex of heart; urine loaded with lithates; not albuminous. Two gallons of fluid drawn off by tapping on Mar. 6. Seven days after, peritonitis, and rapid sinking. P. M.—About Oiv fluid in abdomen; recent lymph on intestines. Liver cirrhose, wt. 3 lbs. 2 oz. Large firm spleen, wt. 18 oz. Kidneys granular. Heart healthy.

Diuretics and stimulants, and operation as above.

*Hepatic Disease.—T. H., æt. 37. Shoemaker. Adm. Jan. 21. Disch., at own request, Mar. 12. In H. 50 days. *Relieved.* Dr. Beale, vol. 5, p. 104. Had syphilis 18 yrs. ago, followed by secondary symptoms. Sudden stabbing pain in right side 1 yr. ago. Six months ago an attack resembling dysentery, followed, after 14 days, by pain and swelling in region of liver, and pain in right shoulder. About two mos. ago a second dysenteric attack. Admitted under Dr. Budd on Dec. 11, and remained in hosp. 19 days. Sallow and wasted. Coppery eruption on skin. Constant pain in right side. Liver extending to 2 in. below umbilicus, and nodulated on surface. Albuminous urine. Constant salivation. Nausea after food. Waterbrash. Constipation.

Hydrochlorate of ammonia for 4 days, and then hydrocyanic acid and bicarbonate of soda. Mercurial ointment rubbed in over liver.

*Gout.—G. P., æt, 31, actor. Adm. Mar. 9. Disch. Mar. 25. In H. 16 days. *Recovery.* Dr. Garrod, vol. 2, p. 70. A free liver. Had two slight attacks of delirium tremens. No hereditary predisposition to gout. First attack in right great toe 9 years ago. Slighter attacks since. Severe general gout a year ago, and every 6 weeks since. Previous illness 9 days. Commenced in right hand, wrist, and elbow; then right foot, left hand, and knees. Slight excess of uric acid in blood serum. Chalk stone on 2nd joint of right fourth finger. Urine not albuminous. No thoracic disease. No pain after 21st day.

Bicarbonate of potass, colchicum and sulphate of magnesia. Afterwards ammoniated tincture of guiacum and iodide of potassium.

*Purpura.—H. C., æt. 21. Light porter. Adm. Jan. 20. *Died* on Feb. 2. In H. 13 days. Dr. Garrod, vol. 2, p. 9. No deficiency of vegetables. Father gouty. Loss of flesh 3 mos. Epistaxis on and off 6 weeks. Purpuric spots on legs 4 weeks. Headache. Gums spongy and bleeding; blood in motions and urine. Systolic bruit over base of heart. Tongue dry and brown. P. 136. Delirium on 12th day after adm. Tenderness on pressure over abdomen, and motions and urine passed under him on 13th day. P. M.—Recent lymph on liver. Pus in peritoneal cavity. Purpuric spots on all the viscera. Spleen friable, wt. 11b. 13 oz. Extensive old disease of aortic valves.

Camphor mixture for 2 days. Then with large doses of sulphate of iron, and for the last 2 days gallic acid. During 2nd week wine 6 oz.; changed for brandy 8 oz. on 11th day.

*Purpura.—M. B., æt. 67. Charwoman. Adm. Jan. 17. *Died* on Jan. 25. In H. 8 days. Dr. Garrod, vol. 2, p. 6. For last 3 mos. lost appetite, and lived chiefly on biscuits and porter. Prev. illness 8 days. General purpuric eruption, commencing on arms, where large patches formed. No soreness or bleeding of gums. Dark motions. Tongue dry and brown. Pulse 100 feeble and irregular. Loud rubbing sound over heart on 2nd day after adm. (7 days). No increase of cardiac dulness. On 5th day after adm. dulness and crepitus over left base. Delirium for 2 days before death. P. M.—Wt. of heart 15 oz; recent lymph on its surface; Left vent. hypertrophied. Lower lobes of lungs firm and sank in water.

Beef tea diet, slice and vegetables, wine 4 oz. and mist. camph. for three days.

Then quinine and opium and wine 8 *oz. Cardiac region painted with iodine. On 7th day sal-volatile and ammonia and brandy* 12 *oz.*

PURPURA.—C. E., æt. 21. Kitchenmaid. Adm. Jan. 19. Disch. Jan. 31. In H. 12 days. *Recovery.* Dr. Beale, vol. 6. p. 76. Previous general health good. No deficiency of vegetable food before adm. Prev. illness 1 week. Purpuric eruption on legs and feet, accompanied by pain and stiffness. No sponginess of gums.

Sesquichloride of iron.

*CHRONIC ECZEMA.—Catherine G., æt. 3. Adm. Feb. 4. Disch. Feb. 28. In H. 24 days. *Relieved.* Dr. Beale, not in case book. Eczema of face and arms. Admitted under Dr. Beale on Sept. 25, 1862. with eczema of face, scalp, neck, arms. and legs; remained in H. 97 days, and was nearly well on discharge.

Glycerine and bicarbonate of potass, grey powder and rhubarb, and local application of nitrate of bismuth ointment.

*CHRONIC ECZEMA.—H. B., æt. 14. Bricklayer's boy. Adm. Jan. 20. Disch. Mar. 14. In H. 53 days. *Relieved.* Dr. Johnson, vol. 16, p. 83. Eczematous eruption on face and scalp 12 years on and off. Abscess below right ear. General health never very good.

Cod liver oil and liq. potass arsenitis (*m* iij *gradually increased to m* vij). *Locally oxide of zinc and glycerine.*

*ECZEMA.—S. C., æt. 20. Kitchenmaid. Adm. Jan. 2. Disch. Mar. 21. In H. 78 days. *Recovery.* Dr. Johnson, vol. 15, p. 45. Prev. illness 3 weeks. An abscess in right forearm, followed by eczema, spreading to face and left arm.

Treated with sesquichloride of iron for nearly a month, and then Fowler's solution. Grey powder and rhubarb. Locally glycerine lotion and bismuth ointment.

*ECZEMA.—Wm. M., æt. 12. Adm. Dec. 5, 1862. Disch. Jan. 24. In H 52 days. *Recovery.* Dr. Johnson, vol. 15, p. 27. Prev. illness 8 mos. Eczema of skin over right popliteal space, forming an oval patch about 12 inches long. Small abscess on inside of thigh.

Sesquichloride of iron. Locally Unq. Bismuth. Nitrat. at first, and afterwards equal parts white precip. ointment and compound lead cerate.

ECZEMA.—M. B., æt. 25. Married. Adm. Mar. 30. Dis. Mar. 31. *Made O. P.* Dr. Beale, not in case book. Eczematous eruption on hands and face (3 months).

Not treated while in hospital.

*J. B., æt. 49. Butcher. Adm. Jan. 28. Disch. Mar. 25. In H. 56 days. *Relieved.* Dr. Garrod, vol. 1, p. 30. Gin drinker. Prev. ill. 1 yr. Frequent loose, pale, slimy stools; containing fat (till Mar. 21) and sometimes blood. Pain after food: waterbrash; occasional vomiting; loss of flesh (6 mos.). Headache (2 mos.). Œdema of legs.

Oxide of silver. Afterwards citrate of iron, and cod liver oil.

*G. B., æt. 41. Silk Weaver. Adm. Jan. 7. Disch. Jan. 22. In H. 15 days. *Relieved.* Dr. Garrod, vol. 1, p. 11. No history of ague. Prev. ill. 4 months. Sudden sharp pain over spleen (4 wks.) followed by a swelling in left hypochondriac and part of left lumbar, umbilical and epigastric regions. Through this the heart's sounds heard distinctly. Six lowest left intercostal spaces filled up. Dulness or percussion over this space, and thickening of skin. His occupation does not cause any undue pressure on this part.

Syrup of iodide of iron, and iodine paint over swelling.

POISONING BY OXALIC ACID.—Mary M'M., æt. 63. Adm. Mar. 9. Disch. Mar. 14. *Recovery.* Dr. Beale. Not in case book. Cup taken away from her before much had been swallowed. Lips and tongue swollen and inflamed.

T. R, æt. 33. Labourer. Adm. Dec. 31, 1862. Disch. Jan. 7. *Recovery.* Dr. Johnson. Not in case book. Half starved. Prev. ill. 14 days. Swelling and redness of legs, with great general depression.
Carbonate of ammonia, chloric ether and bark; wine and nourishing diet.

Debility.—C. S., æt. 38. Needlewoman. Adm. Jan. 13. Disch. Jan. 28. In H. 15 days. *Relieved.* Dr. Johnson, vol. 16, p. 74. Hard worked and ill fed. Always epileptic: fits very frequent of late. Ten yrs. ago epileptic left hemiplegia. Prev. ill. 14 days. Headache and debility.
Quinine and sesquichloride of iron, and morphia at bed time.

Debility.—Maria P., æt. 60. Adm. Jan. 19. Disch. Jan. 31. In H. 12 days. *Relieved.* Dr. Johnson, vol. 15, p. 67. Winter cough 10 yrs. Prev. ill. 4 yrs. Increasing debility and loss of muscular power. No pain or impaired sensation.
Quinine and sesquichloride of iron.

Hysteria.—Catherine B., æt. 27. Servant. Adm. Feb. 17. Disch. Feb. 25. In H. 8 days. *Relieved.* Dr. Johnson, vol. 15, p. 9. Cough and occasional loss of voice and globus hystericus. Catamenia regular. Tenderness over larynx 1 wk. Interior of larynx normal.
Sesquichloride of iron.

Hysteria.—I. H., æt. 16. Servant. Adm. Jan. 8. Disch. Jan. 21. In H. 13 days. *Relieved.* Dr. Johnson, vol. 16, p. 67. Hard worked. Prev. ill. 2 mos. Debility; suppression of catamenia; pain and tenderness in right hypochondrium where there is an indistinct tumour, disappearing under chloroform.
Iron and gentian, and opiate liniment.

Hysteria.—A. B., æt. 24. Servant. Adm. Feb. 26. Disch. Mar. 25. In H. 27 days. *Relieved.* Dr. Garrod, vol. 1, p. 58. Fits accompanied by globus hystericus. Pregnant 4 mos.
Treated with ammonia and chloric ether, and asafœtida.

Hæteria.—Susan H., æt. 26. Servant. Adm. Dec. 30, 1862. Disch. Jan. 3. In hos. 4 days. Dr. Beale, vol, 5, p. 54. *Recovery.* Hysterical dyspnœa.
Treated with sal volatile and valerian.

Croup.—Emma R., æt. 3. Adm. Feb. 1. *Died* on Feb. 2. Brought in at night. Not in case book. Tracheotomy performed.

Hæmatemesis.—M. F., æt. 38. Servant. Adm. Nov. 20, 1862. Disch. Jan. 17. In H. 58 days. *Recovery.* Dr. Budd, vol. 74, p. 106. Intemperate. Catamenia irregular. Vomiting, pain after food, and sour risings 5½ years ago, and twice since. Prev. illness 6 mos Slight hæmatemesis. Pain after food in epigast. and back. Vomiting. Free from pain and vomiting on Jan. 10.
Treated at first with nitrate of bismuth and paregoric. Afterwards with oxide of silver for 3 days. Light diet.

Hæmatemesis.—J. U., æt. 35. "Tight maker." Adm. Feb. 5. Disch. Feb. 20. In H. 15 days. *Recovery.* Dr. Johnson, vol. 16, p. 99. Hæmatemesis (1 qt.) after lifting a heavy weight 2 yrs. ago. Flatulence and sour risings 2 yrs. ago. Prev. illness 3 days. Vomited about 4 oz. of blood. Pain in epigastrium and back. Retching after food. Appetite bad.
Treated at first with turpentine. Then with sesquichloride of iron and chloric ether. Light diet.

Hæmatemesis.—T. H., æt. 31. Labourer. Adm. Jan. 21. Disch. Feb. 4. In H. 14 days. *Recovery.* Dr. Johnsou, vol. 15, p. 73. Small pox 11 years ago. Prev. illness 7 wks. Shivering; pain in right side; cough; vomiting till 10th day after adm. Hæmatemesis and dark motions 6 times in last 5 weeks.

Largest quantity of blood brought up = 0i. Flatulence and constipation. Heart, lungs, and urine normal.

Treated with hydrocyanic acid and bicarbonate of potass and light diet.

Dyspepsia.—A. C., æt. 31. Laundress. Adm. Jan. 20. Disch. Mar. 4. In H. 43 days. *Relieved.* Dr. Johnson, vol. 15. p. 59. Pain across abdomen. Appetite bad. Constipation. Headache.

Treated with purgatives; opium; asafœtida; and then with strychnia.

Dyspepsia.—A. M., æt. 30. Housemaid. Adm. Feb. 18. Disch. Feb. 28. In H. 10 days. *Relieved.* Dr. Johnson, vol. 16, p. 103. Prev. ill. 2 mos. Suppression of catamenia. Pain after food, nausea, vomiting, waterbrash.

Ammonio citrate of iron, soda bicarb., and hydrocyanic acid. Purgatives.

Dyspepsia.—L. L., æt. 30. Needlewoman. Adm. Mar. 3. Disch. Mar. 18. In H. 15 days. *Recovery.* Dr. Beale, vol. 6, p. 111. Pain in epigastrium, and right side. Vomiting. Slight leucorrhœa.

Hydrocyanic acid and bicarbonate of soda, and purgatives.

Dyspepsia.—P. B., æt. 45. Baker. Adm. Feb. 11. Disch. Feb. 25. In H. 14 days. *Recovery.* Dr. Johnson, vol. 15, p. 89. Temperate. No family hist. of cancer. Prev. ill. 14 days. Pain and soreness of epigastrium; vomiting soon after food; waterbrash; constipation.

Bismuth and magnesia, light diet and purgative enema. Afterwards with hydrocyanic acid.

Dyspepsia.—T. H., æt. 39. Railway Guard. Adm. Mar. 16. Disch. Mar. 28. In H. 12 days. *Relieved.* Dr. Johnson, vol. 15, p. 135. Intemperate. Prev. ill. 9 mos. Sour risings after food. Flatulence and pain in epigastrium.

Strychnia and sesquichloride of iron.

Periostitis.—T. F., æt. 38. Labourer. Adm. Dec. 27, 1862. Disch. Jan. 2. *Relieved.* Dr. Beale, vol. 6, p. 57. Secondary syphilitic periostitis, and eruption on scrotum.

Iodide of potassium. Locally, white precipitate ointment.

Exhaustion.—W. J., æt. 32. Labourer. Adm. Feb. 2. Disch. Feb. 7. *Recovery.* Dr. Johnson. Not in case book. Exhaustion from travelling on foot.

Anæmia.—S. B., æt. 22. Nursemaid. Adm. Dec. 20, 1862. Disch. Jan. 28. In H. 39 days. *Recovery.* Dr. Budd, vol. 74, p. 135. Very hysterical. Catamenia regular. Prev. ill. 3 wks. Slight anæmia. Basic systolic bruit over heart, and venous murmur in neck (21 days). Severe pain in right side on Jan. 1 (22 days).

Valerian, sulphate of iron, and asafœtida.

Anæmia.—M. A. L., æt. 20. Servant. Adm. Dec. 3, 1862. Disch. Feb. 3. In H. 62 days. *Recovery.* Dr. Budd, vol. 74, p. 125. Prev. ill. 2½ months. Catamenia irregular. Systolic bruit over base of heart and venous murmur in neck.

Sesquichloride of iron 12 *days, sulphate of iron, and quinine,* 16 *days, and fer reduit,* 34 *days. Aloetic aperients.*

Anæmia.—M. L., æt. 22. Cook. Adm. Jan. 3. Disch. Jan. 21. In H. 18 days. *Recovery.* Dr. Johnson, vol. 15, p. 51. Shortness of breath on exertion, and palpitation, 3 yrs. Scanty catamenia and occasional leucorrhœa, 5 mos. Headache 5 wks. Occasional vomiting 3 wks. Considerable anæmia. Basic systolic bruit, and venous murmur in neck.

Sesquichloride of iron, and sulphate of iron, and ext. gentian.

*Scarlet Fever.—Eliza G., æt. 24. Servant. Adm. Mar. 12. Disch. April 8. In H. 27 days. *Recovery.* Dr. Beale, vol. 5, p. 140. Prev. ill. 4 days. Commenced with shivering, sore throat, and great difficulty of swallowing. Eruption fully out on adm. Cervical glands swollen and painful. Left tonsil and left side of uvula sloughing. Desquamation about 10th day. Severe case.

Liq ammon. acetatis, and chloric ether, 21 days. Then quinine and sesquichloride of iron. Throat swabbed with glycerine and sesquichloride of iron. Wine 4oz.

Scarlet Fever.—J. C., æt. 23. Married. Adm. April 13. Disch. Ap. 27. In H. 14 days. *Recovery.* Dr. Johnson, vol. 16, p. 169. Suckling 12 months. Prev. ill. 6 days. No evidence of contagion. Commenced with sore throat and headache. Eruption on 2nd day. P. 96. Desquamation on 9th day. Was discharged at her own request before desquamation had ceased.

Liq. ammon. acetatis (7 days), sesquichloride of iron. Throat swabbed with equal parts of glycerine and tinct. sesquichloride of iron.

Scarlet Fever.—Eliza T., æt. 16. Servant. Adm. May 14. Disch. June 13. In H. 30 days. *Recovery.* Dr. Johnson, vol. 16, p. 209. Had measles and small-pox. No evidence of contagion. Prev. ill. 2 days. Commenced with vomiting. Eruption on 3rd day. Slight sore throat. P. 140. Desquamation on 7th day. On 9th day wrists red, swelled, tender, and painful, 2 days. Heart unaffected.

Liq. ammon. acetatis and chloric ether (4 days), sesquichloride of iron (2 days), bicarbonate of potash and quinine and opium (5 days). Hot air baths.

*Scarlet Fever.—Dropsy.—Rosina M., æt. 6. Adm. April 13. Died on May. 10. In H. 27 days. Dr. Beale, vol. 5, p. 154. Scarlet fever in same house. Prev. ill. 5 days. Rash fully out on adm. Desquamation about 14th day. On 17th day very fretful, no appetite, constant vomiting, œdema of face and extremities, and hæmaturia. Epistaxis on 27th and 28th days. Pain and tenderness of abdomen on 30th day; no remission of other symptoms. Death on 32nd day, apparently from exhaustion. No P.M.

Chlorate of potash and dilute muriatic acid (10 days), then sp. ammon. arom., liq. ammon. acetatis, chloric ether, and hydrocyanic acid. Purgatives. Brandy. Dry cupping and sinapisms to loins.

*Scarlatina Puerperalis.—S. C., æt. 25. Married. Adm. May 14. Disch. June 14. In H. 31 days. *Recovery.* Dr. Garrod, vol. 1, p. 108. No previous attack. No evidence of contagion. Confined on May 14th. Restlessness and vomiting on 16th. Sore throat on 18th (1st day). Eruption on 19th. On 3rd day P. 132, R. 44. On 4th day P. 120, R 60. Tongue red and dry. Rash fading. Delirious since 2nd night. Lochia and milk suppressed. Abscesses over hips and sacrum. Urine slightly albuminous. On 5th day P. 120, R. 40; less delirium, tongue moister, urine still albuminous. On 6th day, rational; no albumen; desquamation commencing. P. 92. On 9th day lochia natural. Quite well on the 25th day. Child not attacked.

Liq. ammon. acetatis and chloric ether. Brandy up to 12 oz. Throat swabbed with glycerine and tincture of sesquichloride of iron.

*Scarlatina Puerperalis.—E. B., æt. 19. Married. Adm. Mar. 18. Disch. April 11. In H. 28 days. *Recovery.* Dr. Garrod, vol. 1, p. 73. No evidence of contagion. Had scarlatina when a child. Headache on Mar. 14 (1st day). Confined the next day. Sore throat in evening. On 5th day, P. 140, eruption fully out, slight sore throat, milk partially suppressed, lochia healthy. On 8th day, wrists and left knee swollen and painful (4 days), desquamation commencing. Child free from eruption. Died in convulsions soon after birth.

Sulphate of magnesia, liq. ammon. acetatis and decoction of bark, 8 days; liq. cinchonæ and chloric ether, 8 days; and then liq. ammon. acetatis and chloric ether. Brandy 6 oz., increased to 8 oz. on 12th day.

*Erysipelas.—James P., æt. 14 days. Adm. May 13. *Died* on May 17. In H. 4 days. Dr. Garrod, vol. 2, p. 121. No evidence of contagion. Mother healthy. Prev. ill. 2 days. Commenced behind right ear with redness and swelling, which, by the next day, had extended over the back of the neck; and on the 3rd day covered the upper part of the back, forming a patch about the size of the palm of an adult hand, swelled and hard, and of a deep red colour. No vesications and no fluctuation anywhere. Motions rather loose By the 5th day the inflammation had spread over the sternum and between the scapulæ, and by the 6th day covered about one-third of the surface of the body. The child took the breast well throughout. Attacked with severe dyspnœa on the 7th day, and died a few hours afterwards. Death preceded by slight convulsions. P.M.—The portion of the skin that had been inflamed was of a purplish tint. Venous congestion of brain and sinuses. Lungs congested. Other viscera healthy.

Ol. Ricini ʒi. *Collodion and castor oil applied to eruption. Mother placed on full diet, with extra pint of porter.*

*Erysipelas.—J. P., æt. 34, diver. Adm. Ap. 30. Disch. May 9. In H. 9 days. *Recovery.* Dr. Johnson, vol. 17, p. 1. Acute bronchitis 3 months ago. Ailing for 14 days before admission. Prev. ill. 2 days. Erysipelas of face and upper part of neck, commencing under left eye. No albumen in urine. Convalescent about 10th day.

Quinine and sesquichloride of iron mixture, purgatives, and collodion and castor oil locally.

*Erysipelas.—J. F., æt. 23, shoemaker. Adm. May 6. Disch. May 23. In H. 17 days. *Recovery.* Dr. Johnson, vol. 17, p. 15. A similar attack 2 yrs. ago. Prev. ill. 4 days. Shivering and vomiting, followed by erysipelatous inflammation of face and scalp, commencing on right cheek. Vesications on ears. P. 112, R. 32. Slight bronchitis. Tongue thickly furred. Convalescent on 17th day. Severe case.

Quinine and sesquichloride of iron. Purgatives. Collodion and castor oil locally. Brandy 6 *oz.*

*Erysipelas.—J. F., æt. 43, omnibus time-keeper. Adm. May 30. Disch. June 17. In H. 18 days. *Recovery.* Dr. Johnson, vol. 15, p. 211. Intemperate. Acute rheumatism 7 yrs. ago. Prev. ill. 5 days. Commenced with pain in right great toe, extending up leg. Two epileptic fits soon afterwards. Then right leg from knee to ancle became red, painful and swollen. No gout. On adm. he was slightly delirious. Tremors, spectral illusions, feeble pulse. Two patches of vesication had formed, one on the front, and the other on the inner side of leg. Urine ¼th albumen, containing granular and oily casts, and scattered pus. No abnormal heart sounds. Slept well on night of admission. On 11th day, still tremulous. On 14th day, albumen increased, and slight œdema of legs.

Opium. Brandy 6 *oz. Chloric ether and carbonate of ammonia. Sesquichloride of iron and quinine locally; fomentations; and collodion and castor oil.*

Variola.*—Anne W., æt. 37. Adm. May 18. Disch. May 30. In H. 12 days. *Recovery.* Dr. Johnson, vol. 17, p. 37. Prev. ill. 3 days. Slight attack. Convalescent on 13th day.

Quinine and chloric ether. Hot-air bath. Morphia.

Variola.*—Mary Anne J., æt. 15. Adm. June 1. Disch. June 3. In H. 2 days. Dr. Beale.

Variola.*—Eliza A. J., æt. 22. Adm. April 18. Disch. Ap. 19. Dr. Beale.

* These cases of variola were treated in the hospital, because the Small Pox Hospital was too full to receive them.

Febricula.—Mary Ann S., æt. 25, dressmaker. Adm. Mar. 23. Disch. Ap. 4. In H. 12 days. *Recovery.* Dr. Beale.

Liquor ammoniæ, acetatis, and tincture of squills. Afterwards quinine and sesquichloride of iron.

Febricula —Daniel F., æt. 10. Adm. Ap. 2. Disch. Ap. 14. In H. 12 days. *Recovery.* Dr. Johnson, vol. 15, p. 153.

Liquor ammoniæ, acetatis, and camphor mixture.

Febricula —Sophia O., works a sewing-machine. Adm. Ap. 8. Disch. Ap. 22. In H. 14 days *Recovery.* Dr. Beale.

Nitro-muriatic acid, liquor cinchonæ, and chloric ether.

Febricula.—Jane S., æt. 17, servant. Adm. May 5. Disch. May 20. In H. 15 days. *Recovery.* Dr. Johnson, vol. 17, p. 11.

Carbonate of ammonia and chloric ether (10 *days*). *Afterwards quinine and sesquichloride of iron.*

Febricula.—Mary B., æt. 25. fruit-seller. Adm. June 15. Disch. June 24. In H. 9 days. *Recovery.* Dr. Garrod, vol. 1, p. 134.

Calomel and colocynth purge. Camphor mixture.

*Typhus.—R. J., æt. 22, shoemaker. Adm. Mar. 18. Disch. Ap. 15. In H. 28 days. *Recovery.* Dr. Garrod, vol. 1, p. 71. Small-pox 1 mo. ago. Convalescent about 12 days before adm., but still very weak. Access of typhus gradual. No evidence of contagion. Prev. ill. about 12 days. On adm., face flushed; considerable tremor of limbs; very deaf; thick rubeoloid rash on abdomen and extremities. P. 140, R. 28. Tongue dry and brown. Delirious 2 days. No diarrhœa. No albumen in urine. Convalescent about the 15th day after adm.

Nitro muriatic acid liquor cinchonæ, and chloric ether. Afterwards ammonio citrate of iron, and infusion of quassia. Brandy 8 *oz. Increased to* 12 *oz. on 5th day after adm.*

*Typhus.—J. G., æt. 52, lawyer's clerk. Adm. Ap. 15. Died on Ap. 22. In H. 7 days. Dr. Garrod, vol. 1, p. 87. Intemperate. Worked harder than usual during last mo. No evidence of contagion. Access gradual. He gave up work about 6 days before adm. On adm., distinct measly rash on trunk and limbs; tongue coated and dry. P. 112, R. 28. On 5th day after adm, great prostration; delirium; limbs and tongue very tremulous; P. 126, very feeble; R. 36; granular casts, and albumen in urine; an epileptic fit about 1.30 P.M. On 6th day after adm., breathing stertorous. P. 126, R. 36; comatose. Died on 7th day after adm.

Dilute nitro muriatic acid, liquor cinchonæ, and chloric ether. Brandy 6 *oz. on adm. Increased to* 18 *oz. on 19th, and* 24 *oz. on 20th. Tr. opii* mxv *on 20th. Tr. belladonnæ* mxv 4 *tis horis, on 21st.*

*Typhus.—Emma T., æt. 21, married. Adm. Ap. 23. Disch. May 23. In H. 30 days. *Recovery.* Dr. Johnson, vol. 16, p. 183. Suckling for last 12 mos. No evidence of contagion. Access gradual. Prev. ill. about 14 days. Headache; vomiting; pains in back and limbs; and 5 or 6 loose motions a day. Eruption visible, and delirium on evening of 22nd. On adm., copious rubeoloid eruption on trunk and limbs; tongue brownish yellow; P. 124, R. 36; purged 3 times in 24 hours. On Ap. 24, P. 136, R. 40. Delirious on 25th, 26th, and 27th. Convalescent about May 17.

Dilute muriatic acid, quinine, and chloric ether. Then quinine and dilute muriatic acid. Brandy 6 *oz. Increased to* 16 *oz. on April* 27, *and diminished to* 12 *oz. on May* 1.

*Typhus.—T. H., æt. 49, tailor. Adm. May 23. Died on June 5. In H. 13 days. Dr. Garrod. vol. 2, p. 138. Temperate. Subject to gout. First attack 14 years ago. No evidence of contagion. Prev. ill. 11 days. Violent shivering,

loss of appetite, sleeplessness, and constipation. On adm., thick measly rash on trunk and limbs; tongue dry and brown; P. 124, very feeble; slight headache. Delirious for the first time on night before adm. Urine slightly albuminous. On 19th day, P. 88, R. 28. Semi-conscious; evacuations passed under him. Tongue dry and brown: limbs and tongue very tremulous. Was nearly suffocated by some orange peel, on 21st day. During its removal he struggled a good deal, using the limbs of both sides equally well. The coma became more profound, and he died on 24th day. P.M.—Red softening of right corpus striatum. A cyst as large as a small apple in left kidney. Surface of kidney somewhat granular. Gall bladder contracted on 8 or 10 small stones; other viscera healthy.

Quinine, sulphuric acid, and decoction of bark for 9 days. Then carbonate of ammonia and tincture and decoction of bark. Brandy, 12 *oz.*

*Subacute Rheumatism. - Typhus.—Trismus.—W. C., æt. 46. Labourer. Adm. Jan. 28. *Died* on Ap. 12. In H. 74 days. Dr. Beale, vol. v, p. 110.—Temperate. Has lived badly of late. Admitted with subacute rheumatism. Prev. ill, 21 days. Commenced in ankles and knees, then in wrists, elbows, and shoulders. Profuse sweating. No cardiac disease. Free from pain on Feb. 17, the left shoulder remaining somewhat stiff. On Mar. 15 (first day of fever), headache, lassitude, loss of appetite, and pains in limbs. There was a case of typhus in the ward about this time. On 3rd day, thick rubeoloid eruption on trunk and extremities. On 6th day P. 120, R. 24. Convalescent from fever on 15th day. On 16th day the right leg and thigh become painful and œdematous; tongue coated P. 92. Purpuric eruption on legs about 24th day. On 26th day could not separate jaws more than ¼ an inch; masseter and temporal muscles stiff and painful. Right leg as before. On 28th day, trismus much increased; can swallow well. On 29th day, an epileptic fit about 5 A.M.; 24 fits in next 12 hours; rational in the intervals. Died during the last fit. P.M.—Calvarium very thick. Slight opacity of arachnoid. Brain healthy. Firm coagula in right external iliac and femoral veins. Cord not examined.

Acetate of ammonia, bicarbonate of potass, and chloric ether, and quinine and opium. On Mar. 19 *liquor cinchonæ, chloric ether, and dilute nitro-muriatic acid. Brandy up to* 12 *oz. During the trismus, belladonna and Indian hemp and injections of quinine and beef tea.*

*Typhoid Fever.—J. W., æt. 27, Telegraph Clerk. Adm. April 27. Disch. May 30. In H. 33 days. *Recovery.*—Dr. Beale, vol. v., p. 151. No evidence of contagion. No marked shivering. Prev. ill about 14 days. Slight diarrhœa before admission. On adm. P. 92, R. 36. Five or six rose spots on chest and abdomen. Tympanitis (till May 18). Slight cœcal tenderness from May 1 till May 18. Purged about three times every day till May 11. Cough, slight mucopurulent expectoration, crepitation over bases of lungs. Convalescent about May 19th.

Liq. cinchonæ, chloric ether, dilute muriatic acid, sp. ammon, co. and decoction of logwood (21 *days*). *Turpentine stupes. Brandy* 6 *oz. on admission, increased to* 12 *oz. on April* 29, *and* 16 *oz. on May* 2. *Quinine and sesquichloride of iron during convalescence.*

*Typhoid Fever and Pneumonia.—C. P., æt. 29. Hospital Nurse. Adm. April 5. Disch. May 11. In H. 34 days. *Recovery.*—Dr. Garrod, vol. i., p. 84. Had "brain fever" seven years ago. Had scarlatina. Some cases of *typhus* in her ward but no *typhoid*. Prev. ill 3 days. Commenced with shivering. On second day vomiting and purging, pain in head dry cough, and delirium (3 days). Dullness (16 days) and fine crepitation at right base, extending up to angle of scapula on 4th day. On 5th day, P. 112, R. 28. Vomiting on 6th and 7th days. On 10th day some rose spots on back, tongue dry and brown, slight cœcal tenderness (7 days). Abundant rose eruption over abdomen and chest on 12th day.

Purged about 3 times in 24 hours; no blood. Convalescent on 25th day of illness.

Acetate and carbonate of ammonia and chloric ether for a week. Then potash in effervescence. Wine, 4 oz., on adm. changed for brandy, 6 oz. on 15th day (16 days).

*Pneumonia.—H. W., æt. 48. Auctioneer's Porter. Adm. April 3. Disch. May 2. In H. 30 days. *Recovery.* Dr. Johnson, vol. 16, p. 161. Prev. ill. 7 days. Caught cold. Great prostration. Delirium for 4 days after adm. On adm. P. 136., R. 36. Dullness on percussion over left lung behind, from midway between spine and angle of right scapula downwards, with bronchial breathing, bronchophony and diminished vocal vibration. Expectoration viscid, tinged with blood till 18th day. Breathing then bronchial, crepitus heard on a deep inspiration. On 27th day breathing still bronchial about angle of scapula.

Liq. ammoniæ acetatis, and chloric ether (10 days), then quinine and sulphuric acid. Turpentine stupes. Brandy, 6 oz.

*Pneumonia.—W. B., æt. 26. Labourer. Adm. Mar. 4. Disch. April 4. In H. 31 days. *Recovery.* Dr. Beale, vol. v., p. 132. Much exposed to wet and cold. Prev. ill. 4 days. Shivering, pain between shoulders and in right side, cough, and dyspnœa. On adm. dullness on percussion, and bronchial breathing over back of left lung from spine of scapula downwards. Expectoration tinged with blood. On 9th day P. 104, R. 40. Countenance dusky, tongue dry, is much purged. On 33rd day base of left lung still slightly dull; fine crepitus at end of inspiration. No expectoration.

Liq. ammon. acetatis, carbonate of ammonia, and chloric ether (20 days). Brandy, 6 oz., increased to 12 oz. on 8th day. Turpentine stupes. Sesquichloride of iron during convalescence.

*Pneumonia.—C. W., æt. 16. Cigar Maker. Adm. April 10. *Died* on April 14. In H. 4 days. Dr. Beale, not in case book. No history. On adm. face flushed, violent delirium, skin hot, no rash. P. 84, R. 20. Dulness on percussion, crepitation, and bronchial breathing over right base. Left base not examined. Unconsciousness. P. 140. Death. P.M.—Red hepatization of lower lobes of both lungs. Kidneys congested. Brain not examined.

Dilute nitro-muriatic acid, liq. cinchonæ and chloric ether. Brandy 24 ozs. Turpentine stupes.

*Pneumonia.—C. L., æt. 19. Servant. Adm. Mar. 31. Disch. May 20. In H. 50 days. *Recovery.* Dr. Johnson, vol. xvi, p. 159. Syphilis 12 months ago. Prev. ill. 14 days. Got wet. Shivering, headache, and dry cough. On. adm. dulness on percussion over left lung behind from angle of scapula downwards with increased vocal vibration, bronchial breathing and bronchophony, and slight crepitus. P. 108, R. 26. On 16th day friction sound below left mamma and angle of left scapula (5 days). No expectoration throughout.

Carbonate of ammonia and chloric ether. Turpentine stupes to chest. Quinine. Iodide of Potassium. Brandy, 3 ozs. (11 days), then 4 ozs.

*Pneumonia.—J. C., æt. 36. Tailor. Adm. June 9. Disch. June 24. In H. 15 days. *Recovery.* Dr. Garrod, vol. 1, p. 132. For 9 years short-winded and subject to winter cough. Prev. ill. 4 days. Shivering, loss of appetite, pain in right side, cough, and dyspnœa. On 2nd day rusty sputa (11 days). On adm. dulness (14 days), increased vocal vibration, bronchial breathing (4 days), and crepitation over lower lobe of right lung. P. 114, R. 48. Returning crepitation on 7th day. Convalescent about 18th day.

Carbonate of ammonia gr. x. 6tis horis. Calomel and colocynth. Turpentine stupes. Brandy, 6 ozs. for 5 days, then 4 ozs.

*Pneumonia.—J. K., æt. 30. Porter. Adm. Mar. 12. Disch. April. 4. In H. 23 days. *Recovery.* Dr. Beale, vol. 6, p. 117. Prev. ill. 7 days. Commenced with pain under right nipple, and cough. On adm. dulness on percussion, and

fine crepitation over lower two-thirds of lower lobe of right lung. Sputa yellow, transparent and viscid. Chlorides of urine very small in quantity.

Carbonate of ammonia and chloric ether (6 days). Turpentine stupes. Quinine. E.L. to right side on 14th day. Brandy, 4 ozs.

*Pneumonia.—J. C., æt. 41. Labourer. Adm. April 14. Disch. May 20. In H. 36 days. *Recovery.* Dr. Beale, vol. 6, p. 129. Cough all last winter. Prev. ill. 11 days. Shivering, loss of appetite, great prostration, cough, and shortness of breath, hæmoptysis diarrhœa, and vomiting. On adm. P. 108, R. 44. Dulness on percussion, and large crepitation over right side behind from spine of scapula downwards. Expectoration very copious, frothy, and rusty. Bronchial breathing from 14th to 28th day. Large crepitation over base of left lung on 32nd day. Slow convalescence.

Carbonate of ammonia, chloric ether, and liquor ammoniæ acetatis. Brandy, 18 ozs. Turpentine stupes. Sesquichloride of iron during convalescence.

*Pleuro-Pneumonia.—J. A., æt. 20. Compositor. Adm. May 14. Disch. May 27. In H. 13 days. *Recovery.* Dr. Johnson, vol. 16, p. 205. Prev. ill. 8 days. Got wet. Shivering, headache, and loss of appetite, and on 5th day pain in chest, cough, and expectoration of about 0j of blood. On adm. P. 68, R. 24. Dulness on percussion, bronchophony, diminished vocal vibration, and small crepitation over lower lobe of right lung. Expectoration frothy and untinged. No bronchial breathing. On 14th day crepitus over right base looser and more extensive, friction sound at angle of right scapula (8 days). Convalescent about 19th day

Carbonate of ammonia, liq. ammon. acetatis, chloric ether, and quinine, and opium for 6 days. Then quinine, sulphuric acid and chloric ether. Turpentine stupes.

Pleurisy.—Paul M., æt. 17. Employed in a mirror factory. Adm. Mar. 9. Disch. Ap. 20. In H. 42 days. *Recovery.* Dr. Johnson, vol. 15, p. 127. Prev. ill. 4 day. Shivering, headache, cough with slight expectoration pain below left nipple. On adm. dulness over left lung as high as nipple in front, and to midway between spine and angle of scapula behind. Diminished vocal vibration and absence of breathing over dull space, friction sound in front. No œgophony. P. 100, R. 24. Tongue furred. Urine not albuminous. On 8th day dulness more extensive, no friction sound. On 27th day slight dulness at left base, respiratory sounds natural.

Liq. ammon. acetatis, carbonate of ammonia, and chloric ether. E.L. to left side on 13th day. Quinine and sesquichloride of iron during convalescence.

Acute Bronchitis.—C. R., æt. 40. Housemaid. Adm. April 21. Disch. May 9. In H. 18 days. *Recovery.* Dr. Johnson, vol. 16, p. 179. Pleurisy 6 years ago. Prev. ill. 14 days. On adm. crepitation over lower part of lungs behind. Very little constitutional disturbance. Convalescent about 21st day.

Carbonate of ammonia, chloric ether and liq. ammon. acetatis. Turpentine stupes. Quinine and acid.

*Acute Bronchitis.—W. P., æt. 62. Van-driver. Adm. March 23. *Died* on April 4. In H. 12 days. Dr. Beale, vol. vi., p. 124. Frequent attacks of gout during last 20 years. Grandfather gouty, but father not so. Seven months ago a severe attack of diarrhœa, lasting 3 weeks. Loss of appetite, flesh, and strength; vomiting, and sour eructations, 3 months. No abdominal tumour perceptible. On April 1st shivering, sore throat, pains in belly and limbs, loss of appetite. P. 100. Accompanied by severe bronchitis, under which he sank.—P.M.—Lungs emphysematous; tubercles in apices. Bronchial mucous

membrane considerably inflamed. Heart large. Left kidney dilated into cysts.

Compound jalap powders. Pepsine. On April 2, liq. ammoniæ, acetat., aromatic spts. ammonia, and chloric ether. Brandy, 4 oz., and turpentine stupes.

*Acute Bronchitis.—M. K., æt. 23. Servant. Adm. May 12. Disch. June 13. In H. 32 days. *Recovery.* Dr. Garrod, vol. 2, p. 113.—Amenorrhœa 5 months. Prev. ill. 3 days, commenced with shivering and pain in left side. Very low on adm., P. 124, R. 32. Rhonchus and sibilus all over lungs, fine crepitation at bases. Slight dullness over left base. Expectoration frothy and untinged. On 6th day P. 132. R. 36. Dullness over left lung behind, from spine of scapula downwards. Increased vocal vibration, and large crepitation. Expectoration more viscid. Convalescent about 20th day.

Aromatic spts. ammonia, liq. cinch., and chloric ether, afterwards changed for carbonate of ammonia gr x, 6tis horis. Turpentine stupes. Brandy, 6 oz. Quinine during convalescence.

*Acute Bronchitis.—B. L., æt. 16. Smith. Adm. June 1. Disch. June 13. In H. 12 days. *Recovery.* Dr. Beale, vol. 5, p. 176. Short-winded and subject to coughs. Slight cough and expectoration, 5 months. Never spat blood. Prev. ill. 14 days. On adm. high fever; rhonchus and sibilus all over chest; resonance normal. Expectoration scanty and mucilaginous.

Dilute nitric acid and chloric ether. Turpentine stupes.

*Bronchitis.—Bronzed Skin.—E.U., æt. 24. Cook. Adm. May 12. Disch. June 12. In H. 32 days. *Relieved.* Dr. Beale, vol. 6, p. 150. Has had cough and palpitation, and shortness of breath on exertion for some years. Prev. ill. 3 weeks. Pale, weak, and thin. Thyroid gland much enlarged. Brown patches with light interspaces on skin of chest, arms, hips, neck, and cheeks. Appetite bad. Cough and copious mucous expectoration. Crepitation over bases of lungs. R. 32, P. 100. Basic systolic bruit. Venous hum. in neck. Improved.

Sesquichloride of iron, hydrocyanic acid, and glycerine. Wine, 12 oz.

Bronchitis—L. S., 57. Married. Adm. March 5. Disch. May 16. In H. 72 days. *Much relieved.* Dr. Beale, vol. 5, p. 138. Bronchitis, and œdema of ankles; sloughing ulcer on left ankle.

Sesquichloride of iron, and inf. quassiæ. Morphia. Carrot and yeast poultices to ulcer.

Chronic Bronchitis.—C. C., æt 60. Adm. April 27. Disch. May 16. In H. 19 days. *Recovery.* Not reported in case book.

*Emphysema and Bronchitis.—Dropsy.—E. S., æt. 43. Cabman. Adm. May 14. *Died* on June 13. In H. 30 days. Dr. Johnson, vol. 15, p. 185.—Intemperate. D. T. 10 yrs. ago. 7 or 8 attacks of gout. Winter cough 9 yrs. Prev. ill. 1 month. Œdema of legs and ascites, 14 days. Face dusky. Urine scanty and albuminous. Rhonchus and crepitation over lungs. Expectoration copious, purulent, viscid. P. 116, R. 24. Great relief from puncture of legs. P.M.—Lungs adherent, emphysematous, congested. Heart, 18 oz. Right ventricle thick and firm, right auricle dilated, left ventricle flabby, mitral orifice large, valves healthy, kidneys conjested.

Carbonate of ammonia and chloric ether (4 days), then quinine and chloric ether. Turpentine stupes. Brandy. Chlorodyne.

*Emphysema and Bronchitis.—Dropsy.—Purpura.—M. F., æt. 47. Midwife. Adm. April 7. *Died* on April 13.—In H. 6 days. Dr. Johnson, vol. 16, p. 161. Cough and occasional dyspnœa, 10 yrs. Much worse during last 2 yrs. Anasarca 6 wks. Hæmatemesis 14 days ago. Purpuric eruption, chiefly on

back and legs, 1 wk. On adm. face livid and anxious. Considerable dyspnœa. General dropsy. Large purple patch, after gangrenous, on outer and upper part of right thigh. P. 144, R. 40. Rhonchus and sibilus over lungs. Pulsation in scrobiculus. No bruit, urine normal. P.M.—Heart, 17½ oz. Right cavities dilated, atheromatous patches on valves. Upper lobes of lungs adherent, containing scattered chalky masses. Liver everywhere adherent, 3 lbs., fatty.

Aromatic spts. of ammonia and sulphuric ether. Brandy.

Emphysema and Bronchitis.—Dropsy.—S. H., 51. Coach Spring Maker. Adm. June 1. Disch. June 13. In H. 12 days. *Much relieved.* Dr. Johnson, vol. 17, page 45. Not very temperate. Acute rheum. 12 yrs. ago. Rheumatic gout 9 yrs. ago. Winter cough, 4 yrs. Prev. ill about a week. Œdema of legs. Urine slightly albuminous. Cough since last winter. Physical signs of emphysema. Rhonchus and sibilus over lungs. Convalescent from dropsy on 8th day after adm.

Carbonate of ammonia and chloric ether.

Emphysema and Bronchitis.—Dropsy.—J. K., æt. 36. Hatter. Adm. April 16. Disch. May 16. In H., 30 days. *Relieved.* Dr. Beale, vol. 5, p. 131. Always short-winded, and subject to cough; worse in winter. Œdema of legs 2 mos. On adm. general dropsy. Chest emphysematous. Mucous râles over it. Expectoration copious and frothy. Pulsation in scrobiculus, where a systolic bruit is heard. Urine 10 oz. in 24 hours; slightly albuminous, containing granular casts.

Carbonate of ammonia, chloric ether, and compound decoction of broom tops. 4tis horis.

*Emphysema and Bronchitis.—J. W., æt. Adm. May 20. *Died* on June 3. In H. 14 days. Dr. Garrod, vol. 2, p. 134. Subject to winter cough. On May 11 œdema of legs (9 days), after long standing. On adm. face dusky, cough, and profuse expectoration. Rhonchus of sibilus, mixed with crepitation all over lungs. Systolic bruit over apex of heart. Abdomen distended, tympanitic and fluctuating. No albumen in urine. Delirious 5 nights. Heart's action irregular. Death. P.M.—Fluid in abdomen and pleuræ, lungs congested and emphysematous. Aneurism of thoracic aorta as large as an orange, eroding bodies of 9th, 10th, 11th, and 12th dorsal vertebræ. Spleen, 2½ oz. Heart fatty; valves healthy.

Liq. ammon. acetatis, carbonate of ammonia, and chloric ether. Asafœtida.-Bitartrate of potass and jalap powder o. a. m. Brandy, 12 *oz.*

Emphysema and Bronchitis.—H. H., æt. 38. Laundress. Adm. April 29. Disch. June 24. In H. 56 days. *Unrelieved.* Dr. Johnson, vol. 17. p. 8. Long subject to winter cough. Prev. ill. 3 months. Cough and dyspnœa. Chest very resonant. Rhonchus and sibilus. Sputa abundant, purulent. Systolic bruit over heart. No dropsy or scrobicular pulsation. On 8th day after adm. diarrhœa (11 days).

Carbonate of ammonia and chloric ether and senega. Squills. Astringents and opium for diarrhœa. Turpentine stupes.

Emphysema and Bronchitis.—A. S., 54. Commissionaire. Adm. Mar. 28. Disch. May 11. In H. 44 days. *Relieved.* Dr. Johnson, vol. 16, p. 151. Syphilis 30 yrs. ago. Cough and shortness of breath 3 yrs. Physical signs of emphysema. Rhonchus all over chest. Expectoration, copious, muco-purulent. P. 102, R 34. Heart sounds normal. On Ap. 6, sudden numbness of left hand, and absence of pulse in left radial artery for about 6 hours. Circulation restored by friction of arm. On April 10th œdema of left leg, left popliteal vein hard and tense. Was discharged at his own request. Leg still œdematous. Bronchitis improved.

Carbonate of ammonia, chloric ether and senega. Quinine, chloric ether, and dilute sulphuric acid. Acetate of potash. Quinine, sesquichloride of iron, and chloric ether.

Emphysema and Bronchitis.—H. S., æt. 32. Waiter. Adm. Ap. 21. Disch. May 18. In H. 27 days. *Much relieved.* Dr. Johnson, vol. 16. p. 175. Intemperate. Father died of phthisis. Winter cough 10 years. On adm. lips blue, face dusky, breathing difficult, cough and muco-purulent expectoration. Chest barrel-shaped and resonant, crepitation over lungs. Pulsation in scrobiculus. On 6th day after adm. P. 82, R. 28. Improved. Disch. at his own request.

Carbonate of ammonia, chloric ether, and senega (16 days), then quinine, sulphate of zinc, and dilute sulphuric acid. Turpentine stupes. Inhalation of creosote.

Chronic Bronchitis.—J. R., æt. 74. Porter. Adm. Ap. 10. Disch. May 16. In H. 36 days. *Recovery.* Dr. Beale, vol. 6, p. 148. Cough 3½ years. Prev. ill. 2 days. On adm. cough, dyspnœa, mucilaginous and frothy expectoration. Rhonchus and sibilus over chest. Slight hœmoptysis on May 4.

Carbonate of ammonia, chloric ether and senega. Cod liver oil. Turpentine stupes.

Phthisis.—J. P., æt. 40. Street Hawker. Adm. Mar. 11. Disch. Ap. 22. In H. 42 days. *Relieved.* Dr. Johnson, vol. 16, p. 147. Father, mother, brother, and sister died of phthisis. Prev. ill. 4 months. Cough and loss of flesh. Profuse night sweats; occasional vomiting. On adm. dulness on percussion over right apex. Crepitus and prolonged expiration under clavicle and over supra spinous fossa. Rhonchus and sibilus over right base. P. 104, R. 28.

Cod liver oil, quinine, sulphuric acid, paregoric, and chloric ether.

Phthisis.—T. B., æt. 41. Musician. Adm. Mar. 20. Disch. Ap. 8. In H. 19 days. Dr. Beale, vol. 6, p. 121. Seven months ago while playing trombone vomited (?) blood. Since then cough and expectoration, night sweats and loss of flesh. On adm. dulness under left clavicle, inspiratory click, expiration harsh and prolonged.

Quinine, digitalis, and sulphuric acid. Cod-liver oil.

Phthisis.—J. B., æt. 29. House-painter. Adm. Mar. 27. Disch. June 6. In H. 72 days. *Unrelieved.* Dr. Garrod, vol. 2, p. 88. Distinct blue line on gums, colic about 4½ months ago. Father died of phthisis. Prev. ill. 4 mos., cough and loss of flesh, night sweats, slight hœmoptysis. Both lungs dull over upper third, left most so. Tubular breathing under left clavicle, crepitation behind. Sputa purulent. P. 84, R. 24. Urine afterwards albuminous.

Syrup of iodide of iron and cod-liver oil. Afterwards quinine, and sulphate of iron, and sulphuric acid.

Phthisis.—W. C., æt 28. Tailor. Adm. Ap. 9. Disch. June 3. In H. 55 days. *Unrelieved.* Dr. Johnson, vol. 15, p. 197. No hereditary predisposition. Prev. ill. about 10 months, ascribed to his getting wet through. Cough and expectoration, night sweats, loss of flesh. Diarrhœa 5 wks. On adm. crepitus all over right side. Liver tender and enlarged. Trace of albumen in urine. On May 8th dulness on percussion over greater part of left lung in front, here and under right clavicle, large crepitation. Bronchophony under clavicles, loudest on right side. Diarrhœa ceased 2 days ago. Occasional hœmoptysis while in hosp.

Krameria and logwood, then sulphate of copper and opium, and then acetate of lead and opium. Cod-liver oil.

Phthisis.—P. B., æt. 44. Excavator. Adm. Feb. 27. Disch. Ap. 4. In H. 36 days *Relieved.* Dr. Johnson, vol. 15, p. 111. No hereditary predisposition. Pneumonia (right side) 3 years ago. Since then, cough and expectoration, and occasional spitting of blood. Slight hœmoptysis, dyspnœa, and pain in chest 11 days before admission. On adm. dulness on percussion over right apex, and over left back from spine of scapula downwards. Large crepitation over right apex

and left base. Crepitation, tubular breathing, and prolonged expiration under left clavicle. Expectoration copious and purulent. P. 100.

Carbonate of ammonia, and chloric ether, and liq. ammoniæ acetatis (14 *days*). *Then quinine and dilute muriatic acid. Cod-liver oil.*

Phthisis.—Hepatic Disease.—W. O'D., æt. 29. Clerk. Adm. Mar. 16. *Died* on June 11. In H. 87 days. Dr. Garrod, vol. 1, p. 68. Intemperate. In K. C. H. under Dr. Budd from Nov. 29, 1862, to Jan 5. Had been ill a month with lumbar pain, scanty urine, and dropsy of legs and belly. About 25th day after adm., dulness on percussion, and large crepitation over lower $\frac{2}{3}$ of lower lobe of right lung, and greenish viscid expectoration. Constant vomiting. Urine containing oily casts, and $\frac{1}{3}$ albumen. Highest pulse 120; highest resp. 40. Disch. relieved.

Hydrocyanic acid and mist. effervescens, quinine and dilute nitro-muriatic acid, creosote. Brandy 9 ozs.

On re-admission was very weak, P. 128, R. 36, ascites much increased, urine $\frac{1}{2}$ albumen, dulness and large crepitation over the whole of right chest, vomiting almost incessant. Had been getting worse about 11 days before. P.M.—Right lung universally adherent, tubercle scattered through upper lobe, small cavity in back of apex. Left lung emphysematous, containing scattered tubercle.

Carbonate of ammonia, liq ammon. acetatis and ammon.-citrate of iron. Diuretics. Bitartrate of potass and jalap powder. Hydrocyanic acid, and creosote, to check vomiting. Brandy 6 ozs.

Laryngeal Phthisis.—W. S., æt. 17. Gardener. Adm. Ap. 7. Disch. Ap. 16. In H. 9 days. *Slightly relieved.* Dr. Johnson, vol. 15, p. 163. Father died of phthisis. Cough 6 mos. Hœmoptysis 5 mos. ago, followed by gradual loss of voice. Night sweats. Redness of epiglottis and vocal cords. Ulceration of left false vocal cord, also of right false vocal cord to a less extent.

Cod-liver oil, quinine, and sesquichloride of iron.

Laryngeal Ulceration.—Phthisis.—G. H., æt. 23. Law-writer. Adm. Ap. 25. *Died* on June 3. In H. 39 days. Dr. Johnson, vol. 15, p. 175. Had syphilis 5 years ago, no secondary symptoms. Mother died of phthisis. Huskiness 18 mos. Prev. ill. 6 mos. Gradual loss of voice, and dyspnœa. On adm. thin and pale. Epiglottis swollen. True and false cords ulcerated, especially on right side. Dulness on percussion, and feeble breathing over right apex. The expectoration became nummular and fœtid. Dulness under clavicle increased, and breathing became harsh and bronchial. P.M.—Lungs adherent and crammed with turbercles. Cavity as large as an orange, filled with pus at back of right apex. Extensive ulceration of vocal cords.

Iodide of potassium, and liq. hyd. bichlor. Afterwards cod-liver oil, quinine, and chloric ether. Locally, sol. A.N., steam and creosote inhalation. Leeches to throat.

Laryngeal Disease.—J. M., æt. 32. Butcher. Adm. May 13. Disch. June 6. In H. 24 days. *Relieved.* Dr. Johnson, vol. 17, p. 29. Voice husky 5 mos. Enlargement of glands in left side of neck, $2\frac{1}{2}$ yrs. Swelling of left upper vocal cord. Chest sounds normal.

Quinine and sesquichloride of iron. Sol. A.N. to interior of larynx.

Laryngeal Disease.—J. T., æt. 69. Clerk. Adm. May 2. Disch. May 9. In H. 7 days. *Unrelieved.* Dr. Johnson, vol. 17, p. 4. Prev. ill. 10 wks. Occasional loud snoring noise during inspiration, aggravated by speaking. Slight dyspnœa and dysphagia. No loss of voice. Epiglottis much depressed, so that interior of larynx could not be seen with laryngoscope.

Sal volatile, then sulphuric acid; morphia at night.

*Cancer of Epiglottis.—W. H., æt. 64. Street Newspaper-seller. Adm. Mar. 13. Disch. May 27. In H. 75 days. *Unrelieved.* Dr. Johnson, vol. 15, p. 131. No hereditary predisp., and no appearance of cancerous cachexia. Prev. ill. 7 mos. Commenced with difficulty in swallowing solids. Cannot now swallow anything without great difficulty. Epiglottis the size of a walnut, very hard, and ulcerated on surface. While in hospital the tumour became larger and more ulcerated.

Chlorate of potass and glycerine (28 *days*). *Then iodide of potassium and liq. morph. mur.*

Subacute Laryngitis.—J. E., æt. 32. Hair net Maker. Adm. Mar. 20. Disch. Ap. 22. In H. 33 days. *Relieved.* Dr. Johnson, vol. 15, p. 141. Was struck on thyroid cartilage 5 yrs. ago. After 1 wk. complete loss of voice (3 wks.) and huskiness since. Prev. ill. 3 wks. Hoarseness, pain in larynx, and dyspnœa. On adm. complete aphonia, urgent dyspnœa, and pain and tenderness of larynx. Congestion and swelling of mucous membrane over arytenoid cartilages, vocal cords close together and immoveable.

Leeches to throat, chloric ether mixture, steam inhalation. Afterwards sesquichloride of iron and quinine, and inhalation of vaporised solution of tannic acid.

*Pericarditis.—W. B., æt. 28. Clarionet Player. Adm. June 3. *Died* on June 13. In H. 10 days. Dr. Garrod, vol. 2, p. 153. Had dysentery 5 yrs. ago, during voyage home from India. Cough and expectoration, and pain in left shoulder, about 6 wks. Prev. ill. 2 days. Cough and severe dyspnœa. On adm. cardiac dulness slightly increased. Loud friction sound over base of heart. Dulness and fine crepitation over right side behind, up to spine of scapula, rhonchus in front. P. 120, R. 48. Expectoration frothy, untinged throughout. Motions pasty, pale, and very offensive. Appetite bad. Urine albuminous. On 8th day, bronchial breathing up to angle of right scapula. On 11th day, severe pain all over front of chest. Dulness on percussion. Friction sound very loud. Delirium. Death. Highest pulse 148; highest resp. 48. P.M.—About 0j of fluid in right pleura, lower lobes of lungs carnified. Pericardium and contents the size of a pumpkin. Lymph on heart rough and recent. Pericardial sac distended with jelly like lymph. Wt. of heart 19¼ ozs. Abscess the size of an orange in left lobe of liver, its roof formed by attenuated diaphragm and pericardium. No communication with pericardial sac. Ragged ulcers in large intestine and ileum. Kidneys congested.

Quinine and alkaline mixture (6 *days*). *Aromatic spts. ammonia, chloric ether and tinct. cardamom. Co.* (3 *days*). *Carbonate of ammonia, squills and senega. Calomel and opium. Brandy up to* 24 *ozs.*

*Pericarditis.—W. A., æt. 36. Commissionaire. Adm. May 9. *Died* on May 15. In H. 6 days. Dr. Johnson, vol. 17, p. 21. Pain and swelling of knees 3 wks. Prev. ill. 7 days. Commenced with shivering. On adm. P. 92, R. 36. Cardiac dulness slightly increased; faint friction; sound over heart; lungs normal. On May 11, loud friction; sound over heart; fine crepitus at bases of lungs; no dulness; sputa untinged and frothy. P. 108, R. 36. Trace of chlorides in urine. No albumen. On May 13, violent attacks of vomiting after food or medicine. Dulness and crepitus all over back of lungs up to spine of scapula. Delirium during night of 14th and rapid sinking the next morning. P.M.—About a pint of serous fluid in pericardium. Heart and pericardium covered with recent lymph. Fluid in both pleura. Right lung carnified.

Quinine, sulphuric acid, paregoric, and chloric ether (2 *days*). *Carbonate of ammonia, bicarbonate of potass, and quinine and opium. Turpentine stupes. Brandy,* 6 *oz. on adm.,* 12 *oz. on May* 14*th.*

Cardiac Disease.—E. W., æt. 34. Formerly a Soldier. Adm. Ap. 6.

Disch. Ap. 25. In H. 19 days. *Relieved.* Dr. Johnson, vol. 16, p. 163. No acute rheum. Pain in chest, palpitation, and shortness of breath on exertion, 3 years. Temporary left hemiplegia some months before adm Lately, noises in ears, cephalalgia vertigo, and dimness of sight. Heart beating 2 in. below and ½ an inch to left of left nipple. Presystolic bruit. Loudest at apex. P. small, 74.

Sulphuric acid and sesquichloride of iron. Morphia at night. Belladonna plaster.

Cardiac Disease.—J. G., æt. 26. Gardener. Adm. Ap. 23. Disch. May 11. In H. 18 days. *Relieved.* Dr. Johnson, vol. 16, p. 191. Acute rheumatism 6 yrs. ago. Pain in chest; shortness of breath on exertion; palpitation and vertigo, 3 years. Œdema of legs and ascites 2½ yrs. ago (9 wks.). Œdema of legs 1 yr. ago. Prev. ill. 1 wk., with increased œdema of legs. On adm. slight œdema of legs. Heaving impulse; cardiac dulness increased; apex 2 in. below and to left of left nipple. Systolic bruit over apex. Bronchitis while in hosp.

Sesquichloride of iron and chloric ether (11 *days*). *Acetate of potash, ammonio-citrate of iron, and chloric ether* (3 *days*). *Then bicarbonate of potash, chloric ether, and carbonate of ammonia.*

Disease of Heart.—Epilepsy.—H. C., æt. 37. Governess. Adm. May 14. Disch. June 23. In H. 40 days. *Unrelieved.* Dr. Johnson, vol. 15, p. 193. Acute rheumatism 10 yrs. ago. Epileptic about 9 yrs. Catamenia irregular for some time. Anæmic. Systolic bruit loudest over apex; diastolic bruit heard over juncture of 3rd left costal cartilage with sternum. Dulness on percussion over left clavicle, and crepitation below it. Harsh, prolonged expiration below right clavicle. Scanty mucous expectoration. Pulse small. Nine fits while in hospital.

Carbonate of ammonia and chloric ether (21 *days*), *then quinine and sesquichloride of iron. Chloroform inhalation for epileptic fits.*

Cardiac Dropsy.—A. R. 53. Married. Adm. Feb. 5. Disch. Ap. 11. In H. 65 days. *Relieved.* Dr. Garrod, vol. 2, p. 32. Never had acute rheumatism. Palpitation of heart and occasional œdema of legs, 3 yrs. Prev. ill. 3 mos. Œdema of legs. Ulcers just above ankles. Venous pulse in neck. Pulsation in scrobiculus. Harsh diastolic bruit at base of heart. Pulse irregular and intermitting. Increased vocal vibration. Feeble breathing and dullness on percussion over back of right lung from angle of scapula downwards Pain in chest. Headache. Severe attack of palpitation while in hospital. Urine slightly albuminous after Feb. 26.

Carbonate of ammonia, and ammon. citrate of iron. Iron and digitalis, separately and in combination.

*Paraplegia.—M. B., æt. 40. Married. Adm. Ap. 6. *Died* on May 15. In H. 39 days. Dr. Beale, vol. 6, p. 134. Gradual palsy of right leg, 10 years; palsy of left leg, 1 year. On adm. rigidity and contraction of hamstring muscles. No loss of sensation or reflex action. Palsy of bladder and sphincter ani. Sudden sinking on 38th day after adm. P.M.—Much pus and shreds of viscid purulent matter, both outside and inside the dura mater. No disease of the bones. The pus had destroyed the dura mater in various places, and had passed through. It had extended from the lower part of the cord as high up as the cervical region. Mere membranous shreds and much altered cord tissue with pus and the pia mater were all that remained of the lower six inches of the spinal cord. Three or four little scales of hard and, apparently, bony matter were found in the pia mater, corresponding to the disorganised portion of the cord. The origin and cause of the condition were not cleared up by the P.M.

Belladonna (3 *wks*), *iodide of potassium. Wine,* 6 *oz.*

Paraplegia.—H. T., æt. 38. Ship Rigger. Adm. Mar. 16. Disch. June 10. In H. 86 days. *Much relieved.* Dr. Garrod, vol. 2, p. 78. Had syphilis. Paraplegia 7 months ago. Loss of sensation; involuntary evacuations. Complete recovery in 3 weeks. Prev. ill. 2 months. Gradual paraplegia, not becoming complete, preceded by lumbar pain. Sensation impaired. Difficulty in retaining and passing water for 1 month after adm.

Tincture of conium. Sulphate of iron and quinine. Strychnia. Treatment much varied.

Paraplegia.—C. D., æt. 55. *Died* on June 9. In H. 24 days. Dr. Beale, vol. 6, p. 160. Much exposed to weather. Bronchitis 2 months ago. Œdema of legs 5 wks. Loss of motion and sensation from umbilicus downwards. Evacuations passed under him. Urine albuminous.

Iodide of potassium. Afterwards carbonate of ammonia.

Hemiplegia.—J. P., æt. 45. Street Hawker. Adm. Mar. 10. Disch. Ap. 4. In H. 35 days. *Relieved.* Dr. Johnson, vol. 16, p. 139. Had lived badly for some months before adm. Drunk for 2 days. On 3rd day (day bef adm.) sudden giddiness, headache, and loss of vision. Speech thick. Face drawn to the right. Loss of motor power in left arm and leg. Worse up to third day after adm. On discharge motor power and articulation nearly perfect.

Blister to nape of neck. Calomel and colocynth. Iodide of potassium and bark.

Hemiplegia.—B. D., æt. 67. Adm. June 12. *Died* on June 14. In H. 2 days. Dr. Beale, vol. 5, p. 190. Taken ill in street. Semi-comatose on admission. On day after adm. palsy and rigidity of right arm, and rigidity of right leg. Evacuations passed under her. Stertorous breathing. Death on 2nd day. No P.M.

Calomel. Aromatic spirits of ammonia.

Hemiplegia.—T. P., æt. 62. Labourer. Adm. Ap. 17. Disch. Ap. 29. In H. 12 days. *Recovery.* Dr. Johnson, vol. 16, p. 171.—Had been travelling on foot during last 3 weeks. Had lived badly during that time, and had been without food for 24 hours before adm. Fell down in street. On adm. speech thick. Loss of motion, impaired sensation, and no reflex action on left side. Tongue protruded towards left side. Left pupil dilated and insensible to light. Almost well on 3rd day after adm.

Quinine and sulphuric acid. Beef tea and brandy.

Hemiplegia.—T. W., æt. 48. Saddler. Adm Ap. 24. Disch. June 3. In H. 40 days. *Unimproved.* Dr. Garrod, vol. 1, p. 92. Prev. ill. 3 months (1 month after discharge from K.C.H.) Sudden insensibility lasting an hour, and left hemiplegia. On adm. slight motor power in right leg, none in arm. Sensation impaired. Childish. Motions and urine passed under him. Paralysis not improved.

Ammonio-citrate of iron and iodide of potassium. Asafœtida. Purgatives. Seton in neck.

Hemiplegia.—W. L., æt. 23. Sailor. Adm. Mar. 30. *Died* on June 19. In H. 81 days. Dr. Johnson, vol. 16, p. 123. Disch. from K. C. H. 3 wks. ago. Convalescent from *left* hemiplegia. Found insensible 2 days before adm. On recovery slight *right* hemiplegia with loss of sensation. Inability to speak or swallow. Sphincters paralysed. Trismus, can just protrude tip of tongue from mouth. The palsied limbs gradually became flexed, and the muscles very rigid. Trismus continued with slight remissions. Deglutition much improved after 10th day. Strength became much reduced by diarrhœa, and by 80th day he could hardly turn in bed, the head had become drawn back very far, and the muscles of the neck very rigid. Respiration became difficult, and he died apparently from asphyxia.

P.M.—White softening, with oily granule cells in both corpora striata and in pons.

Delirium Tremens.—Meningitis.—G. H., æt. 30. Beadle. Adm. May 28. *Died* on May 29. Dr. Garrod, vol. 1, p. 106. Intemperate. Two or three slight attacks before. Prev. ill. 3 days. Commenced on 3rd day after a drinking bout that had lasted a week, with muscular tremors and sleeplessness. Very noisy and talkative the next day. Spectral illusions on 3rd day. On adm. P. 120, pupils dilated. The delirium increased, he became violent, and died suddenly. P.M.—Slight serous effusion into and opacity of arachnoid, chiefly over anterior lobes. Heart hypertrophied, ventricles full of blood.

Tr. digitalis, ʒvij., *in three doses.*

Facial Palsy.—R. M., æt. 17. Adm. June 7. Disch. June 20. In H. 13 days. *Much relieved.* Dr. Garrod, vol. 2, p. 156. Chorea 15 yrs. ago. Acute rheumatism 10 mos. ago. Slept with the window open on night of 5th. The next morning he could not whistle or close right eye. Mouth drawn to left side. Tongue protruded towards the right. Recovery almost complete.

Blister kept open behind right ear. Syrup of iodide of iron.

*Palsy of 3rd, 4th, 5th, and 6th Cerebral Nerves, Right Side.—M. B., æt. 67. Widow. Adm. Mar. 3. *Died* on Ap. 12. In H. 40 days. Dr. Johnson, vol. 16, p. 133. Pain in back of head and left temple 12 mos. Anæsthesia of right half of face, rigidity of right masseter and temporal muscles, and loss of taste in right half of tongue, 4 mos. Ptosis of right eyelid, palsy of recti and loss of sight of right eye, 6 wks. On adm. loss of sensation in right side of face, right eye motionless, cornea insensible. No action of right temporal and masseter muscles. Pain in right side of face. Twitching of facial muscles. Death preceded by severe pain in head. P.M.—Pus beneath dura mater of inner part of right middle fossa of base of skull. A firm scirrhous growth occupied the right anterior fissure of cavernous sinus and extended into the body of sphenoid, posterior part of orbit and carotid canal. Adjacent bone carious.

Iodide of potassium. Morphia. Locally, sulphate of atropine, belladonna, aconitine ointment.

General Paralysis.—Bronchitis.—C. P., æt. 53. Shopman. Adm. May 16. Disch. May 27. In H. 11 days. *Relieved.* Dr. Beale, vol. 5, p. 162. A free drinker. Winter cough 6 yrs. Four attacks of delirium tremens during last 4 mos. Incontinence of urine, 4 mos. Numbness and loss of power in legs 2 mos. Gradual extension of paralysis to arms. Hands feeble, especially the right. Right pupil smaller than the left. Sibilus over lungs. Frothy expectoration.

Carbonate of ammonia, iodide of potassium, and ammonio-citrate of iron.

Amaurosis.—S. P., æt. 40. Valet. Adm. May 16. Disch. June 3. In H 18 days. Made O.P. Dr. Beale, vol. 5, p. 168. Had syphilis 14 yrs. ago followed by sec. symptoms. Gradual loss of sight of right eye, 5 mos. Left eye affected, 2 mos. Cannot see to write or read. Can distinguish large objects about 20 feet off.

Iodide of potassium. Blisters behind ears.

Delirium Tremens.—E. H., æt. 39. Needlewoman. Adm. June 9. Disch. June 13. In H. 4 days. *Recovery.* Dr. Beale, vol. 6, p. 180. Very intemperate. Drinking for 5 days before adm. Convalescent on day after adm.

Liq. ammon. acetatis and aromatic spirits of ammonia. Tr. Opii. m xxv. h s. s.

Delirium Tremens.—G. S., æt. 58. Adm. May 19. Disch. May 26. In H. 7 days. *Recovery.* Dr. Johnson, vol. 16, p. 213. Very intemperate. Epileptic 13 yrs. Frequent attacks of delirium tremens during last 12 yrs. Had been

drinking for 10 days before adm. Prev. ill. 2 days. Sleeplessness. Spectral illusions. No tremor. Tongue steady. Convalescent on 9th day.
Quinine and sulphuric acid. Opium. Purgatives.

Delirium Tremens.—E. M., æt. 40. Charwoman. Adm. June 23. Disch. June 29. In H. 6 days. *Recovery.* Dr. Beale. Erysipelas in face 6 yrs. ago. Has lately had trouble, and lived badly. Headache and sleeplessness for last few days. Slight tremor. P. 48.
Carbonate of ammonia and chloric ether. Opium. Purgatives.

Delirium Tremens.—B. D., æt. 32. Currier. Adm. Ap. 23. Disch. May 2. In H. 9 days. *Recovery.* Dr. Johnson, vol. 16, 185. Had been drinking for a fortnight. Sleeplessness, 1 wk. Tremors. Slight spectra. Two epileptic fits on 24th. Noisy on 26th and 27th. Slept on night of 27th. Rational on 28th. Free from tremor on 29th. Convalescent on May 1.
Purgatives. Opium. Quinine and acid. Brandy 6 *ozs., and porter* 0j.

Delirium Tremens.—J. H., æt. 52. Adm. Ap. 19. Disch. Ap. 29. In H. 10 days. *Recovery.* Dr. Johnson, vol. 15, p. 169. First attack 15 yrs. ago. Has had several since, the last 9 yrs. ago. Had been drinking 8 days before adm. Prev. ill. 2 days. Sleeplessness. Eyes ferrety. Considerable tremor. Slept on 4th night. Spectra at night till 9th day.
Liq. morph. mur. 3ss o.n. *On 4th day, Pil. Sapon. Co.* gr. x. *statim Tr. Opii.* m.xl *at night. Purgatives. Quinine and sulphuric acid.*

Epilepsy.—M. McM., æt. 65. Stone Sawyer. Adm. May 28. Disch. June 9. In H. 12 days. *Much relieved.* Dr. Johnson, vol. 16, p. 225. Brought in by police. Had fits before. Delirium 3 days. Slight bronchitis.
Opium. Quinine and sesquichloride of iron.

Epilepsy.—D. P., æt. 54. Waiter. Adm. June 24. Disch. June 26. In H. 2 days. *Relieved.* Dr. Garrod, vol. 2, p. 200. Intemperate. First fit on morning of adm., three fits afterwards.
Aromatic spts. of ammonia. Pil. Col. Co.

Epilepsy.— — S., æt. 60. ———. Adm. June 28. Disch. June 29. In H. 1 day. *Relieved.* Dr. Garrod.
Aromatic spts. of ammonia. Castor oil.

Epilepsy.—A. W., æt. 48. Charwoman. Adm. May 18. Disch. May 23. In H. 5 days. *Relieved.* Dr. Beale, vol. 6, p. 156. Epileptic 6 yrs. Had a fit 12 days ago, and a very severe one on morning of adm. Tongue not bitten. Fits less frequent during last 2 yrs.
Sulphate of zinc and hyoscyamus. Pil. Col. Comp.

Epilepsy.—S. C., æt. 24. Married. Adm. June 15. Disch. June 20. In H. 5 days. *Recovery.* Dr. Garrod, vol. 1, p. 140. During last 4½ yrs. has had 4 fits. Fell down in street on evening of adm. Convulsions lasted 6 hours. A slighter attack the next day. Coffee-ground vomiting.
Calomel. Sulphate of iron and sulphuric acid.

Chorea.—S. S., æt. 12. Adm. Feb. 12. Disch. May 30. In H. 90 days. *Recovery.* Dr. Garrod, vol. 1, p. 46. Two former attacks 3 yrs. ago and 11 mos. ago. Prev. ill. 30 days. Loss of motion, and sensation in right arm and leg. Choreic movements affecting the paralysed limbs least. Speech much affected. Tenderness over lower dorsal vertebræ. Could walk alone by 27th day after adm. No fright or cardiac disease.
Ammonio-citrate of iron. Asafœtida. Cod-liver oil and shower-baths after 46th day. Purgatives.

Chorea.—G. J., æt. 14. Errand-boy. Adm. Feb. 2. Disch. May 16. In

H. 102 days. *Relieved.* Dr. Garrod, vol. 2, p. 28. Frightened. Fourteen days afterwards choreic movements began in mouth and left side. The right side became affected towards the beginning of April, and the movements of the left side then became rather less.

Oxide of zinc. Sulphate of iron. Asafœtida. Strychnia. Conium. Quinine and sulphate of iron. Shower-baths.

Convulsions.—T. J., æt. 4. Adm. June 27. Disch. June 29. In H. 2 days. *Recovery.* Dr. Garrod.

Chorea.—E. A., æt. 14. Book-folder. Adm. Mar. 14. Disch. Ap. 29. In H. 44 days. *Recovery.* Dr. Johnson, vol. 15, p. 139 Had chorea 5½ yrs. ago, confined to left side. Speech much affected. Prev. ill. 3 wks. Pain in left leg, followed by choreic movements, then in left arm. Speech and deglutition natural. Right side scarcely at all affected. No fright or cardiac disease.

Quinine and sesquichloride of iron. Shower-baths.

Puerperal Mania.—M. McC., æt. 22. Married. Adm. June 5. Disch. June 16. In H. 11 days. *Unrelieved.* Dr. Beale. Confined 3 wks. ago. Prev. ill. 3 days. Violent delirium. Abscess in right shoulder. After evacuation of matter, the shoulder healed rapidly, and she slept more, but did not improve in other respects.

Carbonate of ammonia and chloric ether (3 days). Then quinine and chloric ether. Liq. morphia mur. Brandy and beef tea.

Dysphagia.—A. S., æt. 54. Tailor. Adm. May 26. Disch. May 30. In H. 4 days. *Recovery.* Dr. Johnson, vol. 15, p. 208. Prev. ill. 6 days.

Dilatation by bougies. Sesquichloride of iron and quinine.

*Cancer of Stomach.—G. R., æt. 46. Jeweller. Adm. Jan. 26. *Died* on Ap. 2. In H. 66 days. Dr. Johnson, vol. 16, p. 85. Prev. ill. 2 mos. Pain in epigastrium, and frequent vomiting after food. Sour risings and flatulence. Rapid wasting. On adm., tumour to right of umbilicus, moveable, painful and tender. Coffee-ground vomiting. P.M.—Cancer, the size of an orange, much ulcerated, involving ⅔ of pylorus. Omentum adherent to stomach. Intestines matted together.

Bismuth, magnesia, and dilute hydrocyanic acid. Opium. Cod liver oil.

Hæmatemesis.—S. S., æt. 21. Married. Adm. May 30. Disch. June 13. In H. 14 days. *Recovery.* Dr. Beale, vol. 5, p. 175. Hæmatemesis 1 qt. about 18 mos. ago. Pain after food 3 wks. Vomited about 1 pt. of clotted blood on morning of adm.

Ice. Gallic acid, dilute sulphuric acid, and tincture of opium. Quinine and sulphate of iron.

Hæmatemesis.—J. N., æt. 65. Married. Adm. May 9. Disch. June 20. In H. 42 days. *Relieved.* Dr. Beale, vol. 5, p. 158. Prev. ill. 1 mo. Pain and soreness of epigastrium. Vomiting. Hæmatemesis. Constipation.

Chloric ether, dilute hydrochloric and hydrocyanic acids. Ipecacuanha and rhubarb. Castor oil and warm water enema.

Hæmatemesis.—E. C., æt. 20. Servant. Adm. Mar. 25. Disch. Ap. 25. In H. 31 days. *Relieved.* Dr. Beale, vol. 5, p. 147. Hard worked of late. Loss of appetite, and occasional vomiting, 7 mos. Catamenia suppressed 4 mos. Hæmatemesis (about 1 qt.) a week ago. Tænia.

Hydrocyanic acid and bicarbonate of soda.

Dyspepsia.—M. A. P., æt. 18. Servant. Adm. Ap. 24. Disch. Ap. 29. In

H. 5 days. Dr. Garrod, vol 2, p. 105. In K. C. H. with similar symptoms about a year ago. Pain after food, nausea, flatulence, sour risings, and loss of appetite. Anæmia.

Sal volatile, ammonio-citrate of iron, and inf. of calumba.

Dyspepsia.—R. E., æt. 26. Artists' Colour-maker. Adm. Mar. 30. Dis. April 8. In H. 9 days. *Recovery.* Dr. Johnson, vol. 15, p. 101. Prev. ill. 9 days. Giddiness, pain in abdomen, and diarrhœa. Blood noticed in motions before adm. On adm. loss of appetite, flatulence, and pain after food.

Quinine and sulphuric acid.

Mesenteric Disease.—A. C., æt 9. Adm. Ap. 6. Disch. May 20. In H. 44 days. *Relieved.* Dr. Johnson, vol. 16, p. 165. Anasarca after scarlatina, 5 years ago. Has had dropsy twice since. Purged 3 or 4 times a day. Abdomen tympanitic and tender. No evidence of ascites. Liver large. Appetite at first bad, afterwards voracious.

Chlorate of potass and tincture of opium. Cod liver oil and syrup of iodide of iron.

English Cholera.—M. S., æt. 29. Charwoman. Adm. June 12. Disch. June 17. In H. 5 days. *Recovery.* Dr. Beale. Prev. ill. 2 days. Vomiting and purging. Pains in limbs. Loss of appetite. Tongue furred.

Castor oil and laudanum. Afterwards sal volatile, chloric ether, and tincture of bark.

Diarrhœa.—G. D., æt. 17. Porter. Adm. May 8. Dis. May 11. In H. 3 days. *Recovery.* Dr. Johnson, not in case book. Attacked suddenly on morning of admission with intense pain in abdomen, vomiting, and purging. When admitted was in a state of collapse One loose motion afterwards. Recovered quickly.

Brandy. Mist. camph.

Diarrhœa.—D. R., 28. Servant. Adm. May 13. Disch. May 23. In H. 10 days. *Recovery.* Dr. Johnson, vol. 17, p. 33. Prev. ill. 3 days. Pain in abdomen, purging and vomiting. P. 120. Tongue coated and tremulous. Sleepless.

Nitro-muriatic acid, liquor cinchonæ, and chloric ether. Liq. morph. Mur. at night.

Constipation.—J. A., æt. 22 Hairdresser. Adm. May 18. Disch. May 30. In H. 12 days. *Recovery.* Dr. Johnson, vol. 15, p. 191. Prev. ill. 2 days. Abdomen painful and tender. Vomiting. Constipation. Dulness on percussion over cæcum. Scybala brought away by injection.

Castor oil and warm water. Enemata. Opium. Leeches to abdomen on 21st. Quinine and sulphuric acid.

Constipation.—E. S., æt. 27. Labourer. Adm. May 8. Disch. May 23. In H. 15 days. *Recovery.* Dr. Johnson, vol. 15, p. 180. A similar attack 3 months before. Constipation. Pain in abdomen. Dulness on percussion, and tenderness over cæcum. Tongue thickly coated. Had been salivated before adm. Convalescent on 18th.

Turpentine stupes. Castor oil and warm water. Enemata. Hirud. vj. *to abdomen on May* 10. *Afterwards quinine and sulphuric acid.*

Constipation.—J. W., æt. 19. Servant. Adm. Mar. 28. Disch. June 25. In H. 89 days. *Recovery.* Dr. Beale, vol. p. Hard worked. An attack of constipation and pain in abdomen last summer. Prev. ill. 8 days. Febrile symptoms soon disappearing. Tongue foul. Bowels obstinately confined. Hysterical.

Colocynth, aloes, and calomel. Castor oil, enemata, croton oil, and colocynth. Sulphate of magnesia and tinct. of jalap. Podophyllin and colocynth. Large doses of sulphate of magnesia. Galvanism.

Gonorrhœal Rheumatism.—J. J., æt. 21. Adm. Ap. 6. Disch. June 10. In H. 65 days. *Relieved.* Dr. Johnson, vol. 15, p. 157. Gonorrhœa 1 wk. before adm. Pain in right hip and right heel.

Iodide of potassium, guaiacum, quinine, and sesquichloride of iron. Blisters to hip.

Rheumatism.—A. C., æt. 23. Blacksmith. Adm. Mar. 2. Disch. Ap. 4. In H. 33 days. *Recovery.* Dr. Johnson, vol. 15, p. 117. Prev. ill. 3 months. Shoulders, elbows, wrists, knees and left ankle affected before adm. Shoulders, knees, and left foot painful on adm. Gonorrhœa.

Iodide of potassium, bicarbonate of potash and bark. Strychnia and sesquichloride of iron.

Acute Rheumatism.—Pericarditis.—Acute Renal Dropsy.—T. T., æt. 16. Light Porter. Adm. Mar. 20. Disch. June 25. In H. 97 days. *Recovery.* Dr. Garrod, vol. 1, p. 75. First attack of acute rheumatism 12 years ago. Has had 4 attacks since. Prev. illness 4 days. Ankles and knees, shoulders, elbows, and hands successively attacked. On adm. right knee, elbow, and hand swelled and painful. Systolic bruit over apex of heart. P. 120, R. 40. On 8th day pain in præcordial region. On 9th day to and fro sound heard over heart. On 12th day dulness and bronchial breathing over right lung behind from angle of scapula downwards. On 15th day dulness, bronchial breathing, and œgophony over lower part of left lung. On 18th day diminished vocal vibration over left side behind, crepitus over left lower lobe, and viscid rusty expectoration. On 20th day, friction sound over base of right lung. On 23rd day, no friction sound audible, crepitus over bases, right base still dull. Expectoration scanty, and almost free from blood. Convalescent from rheumatism about 34th day. Œdema of legs on 31st day. Urine containing blood casts and ¼th albumen. Urine still contained a small quantity of blood on discharge.

Quinine and alkaline mixture (8 *days*). *Bark and carbonate of ammonia* (14 *days*). *Syr. Ferri Iodid* (10 *days*). *Liq. ammon. acetatis* (23 *days*). *Quinine and sulphate of iron* (9 *days*). *Liq. ammon., acetatis, and carbonate of ammonia* (5 *days*). *Then ammoniated tincture of guaiacum. Bitartrate of potass and jalap.*

Acute Rheumatism.—T. H., æt. 19. Labourer. Adm. Ap. 1. Disch. May 6. In H. 35 days. *Recovery.* Dr. Garrod, vol. 1, p. 81. First attack. Prev. ill. 1 mo. Commenced in right knee; then in right knee, ankles, and wrists. Soft systolic bruit over apex of heart. Urine alkaline on adm. (Had been under treatment 3 wks.) On 5th day after adm., pain over heart, and to and fro sound (4 days). Duration of illness about 58 days.

Bicarbonate of potass and quinine. Morphia at night. Iodide of potassium added to mixture on 14th day after adm.

Acute Rheumatism.—C. F., æt. 36. Married. Adm. Mar. 7. Disch. Ap. 4, In H. 28 days. *Recovery.* Dr. Garrod, vol. 2, p. 66. First attack. Prev. ill. 7 days. Left knee and hip, right knee and hip, ankles, shoulders, elbows, and wrists affected in succession. Profuse sweating. Systolic bruit over apex of heart on adm. P. 88. No pain after 25th day.

Quinine and alkaline mixture (5 *days*). *Bark and carbonate of ammonia* (3 *days*). *Then quinine and alkaline mixture with iodide of potassium.*

Acute Rheumatism.—F. D., æt. 23. Butcher. Adm. Ap. 8. Went out on June 2. *Convalescent.* In H. 55 days. Dr. Beale, vol. 6, p. 136. Father, rheumatic. Three former attacks, the last 8 mos. ago. Prev. ill. 3 days. Shivering, headache, loss of appetite, pain and swelling of ankles. Was on the river all the day before. On adm., shoulders, wrists, knuckles, ankles and knees affected. Systolic bruit over apex of heart. P. 120, R. 36. On 6th day, heart's sounds muffled, cardiac dulness increased, severe pain in region of heart. On 16th day,

dullness and friction sound over lower part of right lung behind. No pain in joints after 44th day.

Liq. ammon. acetatis and bicarbonate of potass. Quinine and opium. Brandy. Leeches to præcordia on 11th day. Blisters to knees.

*Acute Rheumatism.—S. T., æt. 20. Servant. Adm. June 1. *Died* on June 11. In H. 10 days. Dr. Garrod, vol. 1, p. 122. First attack. Prev. ill. 3 days. Commenced in ankles. The next day the knees, shoulders, elbows, and hands were affected. On 4th day, joints slightly red, profuse sweating, thick creamy fur on tongue. P. 120, R. 48, to and fro sound over base of heart. On 7th day, to and fro sound over whole cardiac region, becoming less intense at apex after 9th day, but remaining very harsh and loud at base till death. On 11th day, dullness over bases of lungs. Delirious for two hours before death. P.M. —Slight serous effusion into each pleura. Pericardium adherent by recent lymph, easily broken down. Heart 17 ozs. Slight atheromatous deposit at commencement of aorta. Kidneys much congested.

Quinine and alkaline mixture. Brandy. Hirud. vj *to præcordia. Tr. opii* mxx, o. n., *after 11th day.*

*Acute Rheumatism.—M. S., æt. 18. Servant. Adm. June 3 *Died* on June 18. In H. 15 days. Dr. Beale, vol. 5. p. 182. First attack. Prev. ill. 7 days. Ankles, knees, wrists, and right shoulder attacked in succession. On adm., knees much swelled, other joints less so. Thick white fur on tongue. P. 100, R. 32. On 9th day, systolic bruit over heart. Diarrhœa after 9th day. On 18th day, slight dulness over right base. On 20th day, crepitation over right base, tympanitis, joints nearly well. On 23rd day delirium, exhaustion, evacuations passed under her. On 23rd day bronchial breathing over right base. Death. P M.—Base of right lung carnified.

Bicarbonate of potass. chloric ether, and ammonia. Afterwards chloric ether, liq. ammoniæ acetatis and logwood. Brandy up to 24 oz. Stupes to abdomen. E L. nuchæ.

Acute Rheumatism.—H. J., æt. 17. Servant. Adm. May 7. Disch. May 30. In H. 23 days. *Recovery.* Dr. Garrod, vol. 1, p. 98. First attack. Shivering and headache 7 days before adm. Had got wet through the day before. On 4th day the left ankle and shoulder became painful and swelled. On adm. (8th day) profuse sweating, tongue thickly coated, knees, shoulders, ankles, and right hand swelled and painful. Systolic bruit over heart, loudest at apex. P. 106. On 10th day, a friction sound over apex (4 days) No pain after 16th day.

Bicarbonate of potash and quinine (14 *days*). *Then quinine and sulphate of iron. Hirud.* vj *to præcordia on 10th day.*

Acute Rheumatism.—M. A., æt. 26. Servant. Adm. Ap. 29. Disch. May 16. In H. 17 days. *Recovery.* Dr. Garrod, vol. 1, p. 96 First attack. Prev. ill. 7 days. Knees, left foot and hand, and right shoulder swelled and painful. Præcordial pain 3 days. Profuse sweating. Tongue thickly furred. P. 88, R. 36. Basic systolic bruit over heart. Joints quite well on 4th day after adm. Left elbow afterwards painful.

Bicarbonate of potass and quinine (10 *days*). *Then sulphate of iron and quinine.*

Acute Rheumatism (?) Hysteria.—E. J., æt. 18. Servant. Adm. Ap. 15. Disch. May 19. In H. 34 days. *Recovery.* Dr. Garrod, vol. 2 p. 98. Hard worked. Prev. ill. 5 days. Shivering and prostration of strength followed after 2 days by pain and tenderness of ankles, headache, vomiting, and profuse sweating. On 5th day, shoulders, knees, and præcordial region painful and tender. On 7th day, P. 68, R. 36. great tenderness on pressure over præcordial region. On 12th day, no pain or tenderness anywhere. Appears quite well. Tenderness over dorsal vertebræ on 25th day. Herpes zoster on 33rd day.

Bicarbonate of potass and quinine. Asafœtida. Ammonio-citrate of iron and aromatic spts. of ammonia.

[*To be continued.*]

I.—CASES ILLUSTRATING THE FORMATION OF SO-CALLED "FALSE MEMBRANES," IN CONNECTION WITH THE IMMEDIATE COVERINGS OF THE BRAIN; WITH OBSERVATIONS.

II.—CASES OF SCROFULOUS DEPOSIT WITHIN THE SPINAL CANAL; WITH OBSERVATIONS.

III.—CASE OF LACERATION OF THE LOWER PART OF THE RIGHT INTERNAL JUGULAR VEIN, CAUSED BY FRACTURED PORTIONS OF CLAVICLE.

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Continued from page 91, *No. VI., Vol. II.*

Case 16.—"*False Membrane*" *in the Arachnoïd Cavity.*—*Scirrhous Ulceration of the Scalp.*—*Gangrene of the Lung.*

The patient was a woman, Sarah T. aged 46, who was admitted into St. George's Hospital, and died March 16th, 1844.*

History.—She had been suffering, for five years, with ulceration of the scalp; and when admitted into the hospital there was a large fungus-like ulcer of the part, with much sloughing, and exposure of the subjacent bone. Eventually much cough and bloody expectoration came on, and she died from extensive disease of the lungs.

Post-Mortem Examination.—*Thorax.*—Extensive Gangrene of the lung was found.

Cranium.—The scirrhous ulceration of the scalp was excessive, but the bone beneath was not diseased. The brain was natural in texture, though giving out much serum when cut into. There was an unusually large amount of fluid in the subarachnoïdean tissues, and in the lateral ventricles. Covering the left cerebral hemisphere a delicate and thin membrane was found; this was easily detached from the shining transparent arachnoïd beneath, and contained several dark "puncta," evidently owing to the presence of blood. Nothing like "bands" of adhesion were found between the opposed layers of the arachnoïd. No scirrhous ulceration existed in other parts of the body.

* Hospital Post-Mortem Book, 1844, No. 53.

Case 17.—*"False Membrane" in the Arachnoïd Cavity.—Old standing disease of the Corpus Striatum.—Hemiplegia.*

The patient was a woman, Zara S., aged 32, who was admitted into St. George's Hospital, April 29, 1845, and died September 2.*

History.—She had suffered from anasarca for seven months, and had been delivered of a child six weeks previous to admission. At the time of her confinement there was much bleeding, but the anasarca then disappeared. It again came on two weeks later, along with palpitation of, and pain at, the heart, to which, at a previous period she had been subject. When admitted, there was orthopnœa and physical evidence of consolidation of the lung; also symptoms of a weak, dilated heart. There was a slight amount of albumen in the urine, the sp. gr. of which was 1016. On the day after admission she was seized with a "fit," attended by total unconsciousness, general convulsive movements of the body, stertor with foaming at the mouth, and lividity of the face, followed by loss of power of the limbs on the r i g h t side of the body. On the day following she had recovered consciousness, but there was no movement of the right arm and leg; the sensibility of the skin of those parts was slightly affected. The speech was difficult, and the mouth drawn to the left side. She recovered the power of moving the right limbs to a certain extent; but was subsequently affected by much dyspnœa, with lividity of the face and delirium. From this she rallied, and, under the use of diuretics with cantharides and suitable stimulants, almost entirely regained power over the paralysed limbs. The dyspnœa increased, and also the albumen in the urine, to a great degree; she had hœmoptysis before death.

Post-Mortem Examination.—*Thorax.*—There was much fluid in the pleural sacs, and in the substance of the lungs, which were compressed. The heart was thinned as to its walls, and dilated as to its cavities; the mitral orifice was contracted by thickening of its valve-flaps.

Abdomen.—The liver was small and contracted, having a thickened and opaque capsule. The kidneys presented numbers of yellow striæ in their cortical parts. There was ulceration of the cervix uteri.

Cranium.—There was a large amount of serum in the subarachnoïdean tissues, and the arachnoïd membrane, generally, was opaque. The inner surface of the dura-mater, covering both hemispheres of the brain, was lined by a thin false mem-

* See Hospital Post-Mortem Book, 1845, No. 211.

brane of a red tinge. The dura-mater, forming the right portion of the tentorium cerabelli, was lined by false membrane in like manner. This false membrane contained a number of arborescent vessels, especially at the posterior part.

The cerebral ventricles were large, containing clear serous fluid. In the central part of the left corpus striatum there was a yellow spot of about the size of a sixpenny-piece, which was much softened. The brain-tissue immediately surrounding this was also softened, but not discoloured.

Case 18.—*"False Membrane" in the Arachnoïd Cavity.—Slight extravasation of Blood beneath the Arachnoïd Membrane.*

The patient was a man, Thomas B., aged 36, who was brought into St. George's Hospital, July 16, 1845, and died two days afterwards.*

History.—He had drunk very hard, and was admitted with pain in the head, and vertigo. For eight days he had been suffering from difficulty in getting sleep, which was attended by strange fancies; and for three nights, it was stated, that he had had no sleep at all. His illness began with feelings of prostration and pains in the limbs.

On the day of admission both pupils were noticed as being somewhat contracted, but acting well to light, and the face was flushed. The pulse was soft, though quick, and the tongue white in the centre. His manner was peculiarly abrupt and tremulous. On the day after admission he was much quieter, having slept better under the use of salines and hyoscyamus, with "bottled stout," as a drink.

Early on the subsequent day he was violently delirious, having gradually become very excited. Opium was given in the form of tincture (3i doses), but in vain. He rather suddenly became much quieter, began to breath in a very laboured manner and died.

Post-Mortem Examination.—*Cranium.*—The vessels of the scalp were much congested. The brain was firm, its cortical parts shewing many "bloody puncta;" the white parts being pale. The cerebral ventricles were somewhat dilated by slightly blood-tinged fluid. The whole of the parietal arachnoïd surface, covering the left central hemisphere, was invested by a thin tawny-coloured false membrane, easily capable of removal, but attached to the arachnoïd by fine and slender bands of adhesion. Here and there, in the false membrane, were blood-

* See Hospital Post-Mortem Book, 1845.

stained dark patches. The corresponding surface of the dura-mater (so called parietal arachnoïd) was perfectly healthy and smooth. Moreover, a small patch of recently effused blood was found beneath the visceral arachnoïd, covering the upper part of the right cerebral hemisphere, near to the longitudinal sinus.

Thorax and Abdomen.—The heart's chambers were somewhat dilated and the lungs congested; otherwise the organs of these cavities were not unnatural.

Case 19.—*"False Membrane" in the Arachnoïd Cavity.—Rupture of Diaphragm and Spleen.—Fracture of Bones of the Wrists after a fall.*

The patient was a man, James T., aged 24, who was admitted into St. George's Hospital, August 29, and died November 25, 1846.*

History.—He had fallen from a height, alighting upon the hands and fracturing the bones of the wrists. Sudden collapse came on, and this was followed by vomiting of blood, pain in the abdomen, and frequent attacks of syncope. Towards the latter end of life no pain was felt, but a sense of extreme prostration was experienced.

Post-Mortem Examination.—*Thorax and Abdomen.*—It was found that there had been rupture of the abdomen and spleen, and that, in connection therewith, abscesses had formed in the substance of the lungs and in the abdomen. *Cranium.*—A thin layer of fibrinous membrane was found lining the dura-mater over both sides of the brain.

Case 20.—*"False Membrane" in the Arachnoïd Cavity.—Scalp Wound.*

The patient was a man, James W., aged 32, who was admitted into the hospital Nov. 27, and died Dec. 11, 1846.†

History.—He was admitted in consequence of a scalp wound, one inch in length, which did not, however, expose the bone; this was owing to a fall whilst he was intoxicated. Headache followed; and subsequently œdema in the neighbourhood of the wound, which spread to the neck and throat. Delirium preceded death, which occurred whilst he was suddenly rising from his bed.

Post-Mortem Examination.—*Cranium.*—Covering the upper portion of the right cerebral hemisphere was a layer of

* See Hospital Post-Mortem Book, 1846, No. 243.
† See Hospital Post-Mortem Book, 1846, No. 254.

effused blood, and a layer of membrane, of a yellow colour, could be peeled off the dura-mater covering the same (the right) cerebral hemisphere. Also, on the opposite cerebral hemisphere a delicate false membrane was found in the arachnoïd cavity.

The other organs of the body presented nothing worthy of notice.

Case 21.—*"False Membrane" lining the Dura-mater.—Fever.*

The patient was a woman, Sarah T., aged 41, who was admitted into St. George's Hospital, August 2nd, and died on the 18th, 1848.*

History.—She was suffering from petechial fever, and had very decided cerebral symptoms. There was much want of sleep and also delirium.

Post-Mortem Examination.—The brain, on section, gave out a large quantity of serous fluid, which was abundant in the sub-arachnoïdean tissues and in the ventricles. On both sides of the brain the dura-mater was lined by a layer of soft membrane, evidently of recent formation, which had produced several adhesions between the opposed serous layers, chiefly at the upper and back parts of the brain.

Case 22.—*Membranous Layer of Blood in the Arachnoïd Cavity.*

The patient was a lady, Jessie D., the widow of a physician, who was brought into St. George's Hospital, with cholera, July 16, 1849, and died on the following day.†

History.—She was almost destitute, and, a few weeks before her admission, had sustained a severe blow on the head. For a month before admission she had had diarrhœa and sickness, and, at times, attacks of severe vomiting. She walked to the hospital, a distance of three or four miles, and when she arrived, the skin of her face was dusky, and the whole surface of the body cold. The pulse was about 85 per minute, but so feeble as scarcely to be perceptible. The breath was cool, and there was great thirst. The tongue was also quite cool, and much coated. The patient was, however, sensible, and complained much of want of sleep. Characteristic cholera-stools were passed by the bowels, and, in spite of treatment, she died.

Post-Mortem Examination.—*Abdomen.*—The glands of Peyer, in the intestines, were found to be unusually large, but nothing further of note was observed about the abdominal organs.

* See Hospital Post-Mortem Book, 1848, No. 176.
† See Hospital Post-Mortem Book, 1849, No. 149.

Cranium.—The brain-substance was rather "wet" on section, but, otherwise, presented nothing worthy of comment; over the cerebral hemisphere, however, on the right side, a thin lamina, or membrane, was found attached to, but easily removable from, the inner surface of the dura-mater. This was evidently formed of a layer of extravasated blood, and was tolerably recent; nevertheless, the colouring matter was beginning to assume a change, and the layer of blood was becoming transformed into a regular membrane.

No indications of injury from without, to the scalp or to cranial bones, was observed.

Case 23.—*"False Membrane" in the Arachnoïd Cavity.—Phthisis.*

The patient was a man, C. N., aged 21, who was brought into St. George's Hospital, February 24, and died March 9, 1858*

History.—He was admitted with pleurisy and phthisis. There were never any symptoms referable to the brain.

On Post-Mortem Examination, tubercles in the lungs and the results of old and recent pleurisy were found. The brain, itself, was healthy; but a thin layer of "false membrane" was met with lining the free surface of the dura-mater.

Case 24.—*Layer of Dense Membraneous Material, containing a cavity, attached to the inner surface of the Dura-mater.—Softening of the Central parts of the Brain.—Epilepsy.—Hemiplegia.—Tetanoïd Spasm.*

The patient was a woman, Mary H., aged 42, who was brought into St. George's Hospital, August 9th, 1850, and died on the second day following.†

History.—Five years previously she had been the subject of epileptic attacks, and, after one such attack, had lost the use of the left arm and leg. The leg had recovered to a considerable degree since, but the arm remained almost entirely without power of movement. Four days before admission she had a succession of from 25 to 30 severe convulsive attacks, and became unconscious. Subsequently muscular twitching came on in the limbs, and she was brought into the hospital partially, but not wholly, unconscious. The whole of the left side of the

* See Hospital Post-Mortem Book, 1858, No. —?

† See Hospital Post-Mortem Book, 1850, No. 144.

body had lost all power of movement, and she was unable to speak or even to protrude the tongue. The face was flushed, the surface generally rather hot, and the pulse 120 per minute.

The bowels were well relieved by injections, and a blister applied to the nape of the neck.

On the day following she was rather more conscious, but the muscles at the back of the arms and legs were affected by clonic spasm, by which the body was thrown backwards in a succession of short tetanic paroxysms. Ice was applied to her head, and blood taken from the arm. Croton and castor oil were also administered, but no benefit followed treatment. Convulsions became more severe; the face more flushed; the evacuations were passed involuntarily; and coma came on before death.

Post-Mortem Examination.—*Thorax.*—The heart was soft in texture and flabby. The lungs were congested. *Abdomen.*—The kidneys were congested. *Cranium and spine.*—The spinal cord was congested generally. There was found extensive softening of the right corpus striatum, of the right optic thalamus, of the back of the fornix, of the under part of the corpus callosum, and of the central part of the posterior lobe of the right cerebral hemisphere. Moreover, on the right side of the brain, and corresponding to the right parietal eminence of the skull, a patch of yellow material, laminated in character, was found adherent to the inner surface of the dura-mater. This was of about the size of a crown-piece, and contained a kind of cavity, in which was a substance looking like silvery scales to the naked eye. This laminated substance could, without much difficulty, be peeled from the dura-mater, to which, however, it was somewhat firmly united. It was also, though very slightly, adherent to the visceral arachnoïd membrane corresponding. The arachnoïd membrane generally, over the right cerebral hemisphere, was more vascular than on the opposite side. There was a slight increase of the sub-arachnoïdean fluid, and the cerebral convolutions were somewhat flattened.

The cortical parts of the brain, generally, and also the cerebellum were very vascular.

[To be continued.]

II.—CASES OF SCROFULOUS DEPOSIT WITHIN THE SPINAL CANAL.

In this communication I propose to offer a series of cases, which have presented themselves during recent years at St. George's Hospital, showing the presence of scrofulous material within the spinal cavity, and illustrating the evil effects which are, for the most part, produced by such deposit upon the spinal cord. Many of the cases are associated with strumous disease of the vertebral elements; others, on the contrary, are not so associated. Again, it will be seen, that the effects upon the enclosed spinal cord vary very much, owing to the locality affected, the amount of scrofulous material deposited, its consistency, &c., &c. As I shall have the opportunity of relating several of these cases, I shall detail them in succession, and postpone such comments and inferences as may naturally arise from their consideration, until the whole have been related.

Case 1.—*Scrofulous deposit within the Theca Vertebralis.—Softening of the Spinal Cord.—Caries of the Vertebræ.—Hepatization of Lung.—Paralysis of the Legs and Arms, &c.*

The patient was a young man, Robert Glade, aged 20, who came under my care, at the St. George's and St. James's Dispensary, July 31st, 1856.

History.—The patient was one of a delicate family, some of whom had died of phthisis. His mother was hump-backed, owing, as it was said, to an injury to the back soon after the birth of the patient. He had experienced no blow or fall, and no severe illness in his lifetime; but, on the 11th of May, 1856, owing to pain in the right shoulder and a so-called "stiff neck" (which was thought to be, possibly, owing to rheumatism), from which he had suffered for the space of a month; he was obliged to give up his ordinary daily employment. Afterwards, he described the pain as going more into the neck and down the back, and as being much aggravated by coughing, or sneezing, or shaking himself, or when dressing, and when lying in bed he was quite unable to get up unassisted, owing to pain in the neck. His mother said he had latterly been "very nervous," and his memory had become somewhat defective. The hands were said to be generally cold, but not so the feet. Moreover, for one month there had been some difficulty in swallowing large morsels. Occasional headache was complained of, and at times vertigo, especially if the bowels were confined.

When first seen at the Dispensary, he was thin and spare, with cold hands, and in walking he supported the chin with his hand; he was also unable to approximate the chin to the breast owing to great and painful stiffness at the back of the neck. There was general tenderness on pressing the neck, which towards the lower part was somewhat full; and the right sterno-cleido mastoïd muscle was more tense and "taught" than its fellow. He complained of much "weakness" in both arms, but especially in the right one, so that he had a difficulty in lifting up anything of even very slight weight, or even to help himself at his meals; and the grasp with both hands, but especially with the right hand was very feeble. The legs were also weak in muscular power, and, at times, in walking, would "give way" at the knees; the right knee was too much inclined outwards. He always walked as if "with the greatest care." There was no cough, and there had been no hœmoptysis. He was wont to have palpitation of the heart, and the pulse was feeble: perspiration was apt to be abundant. Physical examination of the lungs and heart showed nothing wrong. There was no anasarca. The tongue was clean, the appetite good, but the bowels were often confined. The urine was free from albumen. The muscles of the face and also the pupils of the eyes were in a natural condition. The voice was unaffected.

On stripping the boy, it was found that there was curvature of the upper part of the dorsal region of the spinal column towards the right side; and great tenderness was produced by pressure at the middle of the dorsal as well as at the lower of the cervical part (as before mentioned) of the spinal column.

The treatment first adopted consisted in the application of the ung: antimon. potass tart. producing eruptions, about the spine, night and morning, and the use of quinine and cod liver oil, with occasional aperients. It was stated, August 12th, that the legs were weaker.

August 16th, the neck was described as being easier. He "could walk without holding up the chin," and there was no dysphagia. The legs were, however, getting more feeble, and both legs and thighs were stated (especially the right one) to "feel numb" when touched: this was the case also with the abdomen, as high up as the umbilicus. No starting or convulsive movements of the limbs existed, but often, on walking, the legs would "start out too far," and he would "take a longer step" than he expected. The legs were also described as often feeling "dead" in walking, especially the right one, and had of late been cold. The hands were cold, but there was no want of

sensibility of their surface. The ointment and medicines were continued. August 23rd, the legs were said to be much worse as regards numbness and want of sensibility, but he was "much better in himself," and stouter: he could raise himself in bed, and hold up his head better. There had been no spasmodic action of any of the limbs. The appetite was improved. He however complained of feeling pain occasionally across the chest, but there was no cough. The eruptions on the back had still not healed. Quinine and cod-liver oil were continued, and the iodide of potassium, with extract of gentian, given every night in addition.

September 6th, much the same on the whole. Pain was much complained of in the "l e f t" shoulder-blade and arm; and also low down on the left side, above the crest of the ilium, which part was also painful on pressure; but no swelling existed at this region. He continued to pursue the same treatment until September 17th, when I got him admitted into St. George's Hospital, as the power in the legs had become so much more impaired. There was at that time decidedly less pain in the cervical region, even on pressure, but movement of the head in a lateral direction had become difficult. Blisters were, however, applied to the neck with decided relief, and later on a seton was inserted. The surgeon who was asked to examine the vertebræ could find no actual evidence of diseased bone, and the patient was placed under the influence, first of colchicum, and subsequently of the ammonio-citrate of iron. Under the latter he improved for a time, and became able to walk about the room; but the improvement did not last long, as at the beginning of December he was himself quite unable to extend his legs in bed, which, if extended by another, would, as a rule, gradually, though often quickly, become drawn up and flexed more or less on to the belly, the hamstring muscles becoming quite rigid. The sensibility of the skin of both legs, especially of the right one, was very deficient, and much reflex action of both legs could readily be produced by tickling the soles. It was also noticed that the left great toe was always extended, and frequent erections of the penis were complained of. Muscular power in the arms became more diminished. Towards the end of February, 1857, severe dyspnœa came on, and also some degree of cough; the paralysis both of arms and fingers becoming more complete. There was always more complete and decided "kicking up" (as the patient termed it) of the legs. The patient gradually sank; but three days before death had a severe attack of tightness across the chest and urgent dyspnœa, though very little expectoration. He however remained sensible to

the last, and no spasmodic movement of any kind came on before death, which occurred March 4th.

Post-Mortem Examination.—The body was somewhat emaciated. The spinous process of the last cervical vertebra was noticed as being very prominent.

Cranium.—The only thing noticeable about the cranial contents was that the cerebral veins were very turgid.

Spinal Column.—The posterior surfaces of the bodies of the last cervical and the three upper dorsal vertebræ were deprived of all ligamentous or fibrous covering, and the cartilage intervening between these vertebræ was almost quite destroyed, the opposed surfaces being carious. The anterior surfaces of the bodies of the five upper dorsal vertebræ were quite eroded and bathed in thick pultaceous white fluid and pus. It was also noticed that the yellow elastic tissue between the laminæ of the last cervical and first dorsal vertebræ was very remarkably softened and discoloured. On opening the spinal canal a large quantity of purulent fluid and of solid scrofulous deposit was found within the theca vertebralis, opposite the last cervical and first dorsal vertebræ, and this was especially so on the "left" side of the cord. By this deposit, moreover, the out-going roots of the spinal nerves on both sides, but chiefly those on the "left" side were closely surrounded by this deposit, which was very firmly adherent to the dura-mater at the posterior part of the canal. On examining the spinal cord itself, it was found that at the lower part of the cervical and upper portion of the dorsal region it was exceedingly softened, being, opposite the seventh cervical and the first dorsal vertebræ, almost diffluent.

Thorax.—The lungs, generally, were œdematous, and the lower portion of the right lung was hepatized. Much mucus existed in the bronchial tubes. The pleural cavities were natural, excepting that they were diminished by the bulging forward of the bag of pultaceous matter covering the anterior surfaces of the vertebræ.

Case 2.—*Scrofulous deposit occupying a part of the dorsal region of the Spinal Cord, which was also softened.*

The patient was a woman, Elizabeth D., aged 60, who was admitted into St. George's Hospital, August 3, 1831.

History.—For about eight weeks she had been complaining of pain in the back, dribbling away of the urine, and numbness of the legs. A few days after her symptoms first showed themselves there was a want of power in moving the right leg, and subsequently in moving the left leg; and afterwards the

sphincters lost their power, and the integuments of the back began to slough. She sank, and died August 21. Before admission into the Hospital she had been treated with strychnine, and been cupped and blistered. Spasmodic twitchings in the limbs, and a pricking sensation had been produced by the medicine, but no good results.

On Post-Mortem Examination, the lower part of the dorsal region of the spinal cord was found to be softened, and opposite the eleventh dorsal vertebra a mass of scrofulous deposit was found in the substance of the cord. The right half of the chord was more occupied than the left, but at no part was the deposit visible on the external surface of the cord.*

Case 3.—*Scrofulous material adherent to the outer surface of the Spinal Dura-Mater.—Spinal Cord healthy.*

The patient was a man, George P., aged 21, who was brought to St. George's Hospital, April 1, 1846.

History.—He had enjoyed general good health; but about two months previously he had experienced a feeling of constriction and pain across the chest, with a "shooting" to the back, which was followed in six weeks' time by a loss of sensation and of power of motion in the lower limbs, attended by a sense of coldness and numbness and by occasional twitchings in those limbs. When admitted, the general health was good, but there was pain between the shoulders when the vertebræ in that part were pressed, though no irregularity of the spinous processes, &c., existed; no headache was complained of; and there had never been any loss of consciousness. The bowels were much confined, and the motions, and the urine which was not albuminous, were voided with difficulty. After a time all the symptoms increased in intensity; and, by degrees, the arms, to a certain extent, lost sensation and power of motion. The urine also became alkaline and irritating. Perspiration of the skin was profuse about the head, neck, and shoulders; but below the latter the skin was always very dry. Great difficulty in breathing and expectoration of purulent matter came on, and bed sores formed before death, which occurred July 2nd.

Post-Mortem Examination.—Extensive grey hepatization of parts of the left lung, and much mucus in the bronchial tubes were met with, as also softened scrofulous deposit in the bronchial glands. A quantity of scrofulous material was found amidst the

* The preparation exists in St. George's Hospital Museum, Series viii, No. 140.

deep muscles of the back and around the 2nd, 3rd, and 4th dorsal vertebræ and the ribs, &c., which on their anterior surfaces were carious, and this deposit was continuous (by means of two of the intervertebral foramina, which were excessively enlarged) with a quantity of concrete deposit of the same nature within the spinal canal and adherent to the outer surfase of the dura-mater (but confined to the posterior part) of the dorsal region.* The spinal cord was at this part somewhat compressed, and was also softened by the pressure of the scrofulous material. The other parts of the spinal column, spinal cord, and membranes were healthy. The inner surface of the bladder was in a state of chronic inflammation. Moreover, the soft parts about the great trochanter, which was exposed by ulceration, contained an irregular deposit of new bone, quite apart from the original osseous structure of the trochanter.

Case 4.—*Scrofulous material occupying the Spinal Canal.—Pressure upon, and softening of, the Spinal Cord.—Caries of Vertebræ.*

The patient was a man, Michael M., æt. 33, who was admitted into St. George's Hospital, December 2, 1857, having previously been in the Charing Cross Hospital, and subsequently in the Middlesex Hospital.†

History.—He had had pain between the shoulders for 7 or 8 years, and for one year had noticed a projection at the "lower part of the back." Subsequently he lost all power of movement in the legs, and became an out-patient at Charing Cross, where counter-irritation was applied to the region of the curvature, which had the effect of diminishing the pain. The curvature increased, however, and a painful feeling of constriction around the abdomen was experienced, the walls of the abdomen being prominent and tense. Whilst in the Hospital a painful cough came on, and "tubular breathing" at the apices of both lungs was found on stethoscopical examination, along with dulness on percussion. Tonics with wine and good nourishment were given. The paralysis became more complete, and before death wandering of the mind came on. He gradually sank, and died January 30, 1858.

* See preparation in St. George's Hospital Museum. Series viii, No. 142.

† Mr. Shaw, of the Middlesex Hospital, in a note, dated February 6, 1858, informed me that when he saw this patient in that hospital, there was a "perceptible, but not great, projection of the spinous processes of the vertebræ in the dorsal region; and," he says, "I concluded that there was caries. The treatment consisted in painting the affected part with a solution of iodine, and giving tonics, with rest in the recumbent position."

Post-Mortem Examination.—*Thorax.*—The apices of both lungs contained masses of crude tubercle and vomicæ; numerous old pleuritic adhesions existed.

Cranium.—The bones of this cavity were natural. There was much soft fibrine among the sub-arachnoïdean tissues at the base of the brain, corresponding to the so-called circle of Willis, and especially around the optic nerves. The surfaces of the cerebral convolutions were flattened and dry, and the lateral ventricles were much distended with limpid fluid. The central parts of the brain, specially the septum of the ventricles, were much softened, but the fornix was not so. The anterior parts of the thalami optici were decidedly softened; and the vessels at the base of the brain, which to the naked eye appeared natural, were found by the microscope to contain numbers of fat globules within their parietes. *Spinal column.*—The bodies of the three lower dorsal vertebræ were found to be surrounded by a large mass of strumous material of about the consistence of putty, bound down by thickened pleura and sub-pleural tissue, and of this a quantity communicated with the interior of the spinal canal compressing the enclosed spinal cord, which at this part was considerably softened. The outer surfaces of the dorsal vertebræ, which were surrounded by scrofulous material, were much absorbed.

Abdomen.—The viscera of this cavity were natural.

Case 5.—*Scrofulous Abscess communicating with the interior of the Spinal Canal.—The Spinal Cord most probably pressed upon, but not softened.*

The patient was a woman, Mary B., aged 40, and was brought into St. George's Hospital, April 8, 1850.

History.—She had been an inmate of the hospital four years previously, owing to a fall on the back which produced very great pain, but no paralytic symptoms. Of this she recovered, and went on well until three months before her second admission, when she experienced much pain in her back, and had much cough. About eleven days before admission she quite suddenly lost power in both her legs, and when she came in there was evidently lateral curvature of the upper dorsal vertebræ, which were very painful. The stools became passed involuntarily, and a slough formed on the back exposing the sacrum; she sank and died, apparently from the extreme exhaustion owing to the discharge from the slough.

Post-Mortem Examination.—The lungs were found to contain a few old seams of scrofulous deposit, and the opposed layers

of pericardium were throughout adherent. In "the posterior mediastinum" and in front of several dorsal vertebræ (from the 3rd to the 8th), which were carious and softened on their anterior surfaces, was a large scrofulous abscess. The greater part of the body of the 7th dorsal vertebra was absorbed, and by reason also of the absorption of the cartilage between the 3rd and 4th dorsal vertebræ the abscess communicated with the spinal canal. Moreover, the lower part of the body of the 7th dorsal vertebra was tilted upwards and backwards, and, most probably, somewhat pressed upon, but had not softened or otherwise injured, the anterior columns of the spinal cord. The kidneys were congested. The other organs of the body were healthy.

Case 6.—*Scrofulous deposit within the Spinal Cavity and adherent to the outer surface of the Dura-Mater.—Spinal Cord pressed upon, but not softened.*

The patient was a boy, George S., aged 15, who was brought into St. George's Hospital, December 18, 1842.

History.—When admitted he complained of great pain at the back part of the neck, which was increased on motion, and he could not support the head when moved, without supporting it by the hands. There was numbness of the fingers, which increased; and soon afterwards the upper extremities became paralysed. Subsequently the lower limbs became also gradually paralysed, and incontinence of urine came on. Bed sores supervened, and before death, which took place October 14th, much difficulty in swallowing, and dysphagia, occurred.

Post-Mortem Examination.—There was extensive caries of several of the vertebræ, and also of the bones forming the base of the skull; and, corresponding to the 2nd, 3rd, and 4th dorsal vertebræ, was a quantity of scrofulous material, which was firmly adherent to the outer surface of the spinal dura-mater, and extended upwards, through the foramen-magnum, into the cranial cavity. It was found that the spinal cord was pressed upon, though not softened by, the deposit, which however was very firm and solid.* The other viscera of the body were free from disease.

* The preparation exists in the St. George's Hospital Museum, Series viii, No. 141.

Case 7.—*Scrofulous deposit occupying the cervical region of the Spinal Cord.—Similar deposit in the Brain.—Loss of power in the legs, arms, and sphincters, &c.*

The patient was a boy, Michael K., aged 13, who was brought into the hospital, May 2, 1849.

History.—He had for some weeks experienced a gradual loss of power in moving the legs and arms, and subsequently he had completely lost all control over the sphincters of the bladder and bowel. When admitted, much pain in the neck, increased on pressure, was complained of, and pain was produced by any movement of the head backward, but not by any lateral movement. There was also some degree of priapism. When the arms were extended by another person he could raise them, but he could not himself extend them. At a later period, the arms became firmly and permanently crossed over the chest, and great pain was occasioned by any attempt on the part of others to remove them; still later, rigidity of the sterno-cleido-mastoïd muscles on the right side came on; and for some days before death diarrhœa and bed-sores were established. Counter-irritation to the nape of the neck, in addition to other remedies, proved utterly ineffectual in affording relief, and he died September 10th, *the fæces having been observed to pass by the bladder.*

Post-Mortem Examination.—The lower part of the cervical region of the spinal cord (opposite the 5th, 6th, and 7th vertebræ) was much enlarged, and was occupied and rendered very solid by yellow scrofulous deposit in its entire thickness; the dura-mater and pia-mater corresponding to this portion of the cord were thickened and adherent to it.* Above the deposit, the spinal cord was very vascular, and also softened; but lower down it appeared healthy. A large mass of scrofulous deposit, of the size of a walnut, was moreover met with in the right optic thalamus, encroaching on the cavity of the third ventricle, and occupying a part of the substance of the left optic thalamus. The brain generally was rather softened and less vascular than natural; there was only a slight amount of sub-arachnoïdean fluid.

Thorax.—A slight amount of recently-deposited lymph was met with in the pleural sacs, and a small mass of cretaceous material surrounded by a fibrous capsule was found in one lung. In the left side of the pelvis was a sloughy cavity communicating with a slough at the back of the sacrum. This

* The preparation exists in St. George's Hospital Museum, Series viii, No. 139.

sloughy cavity also communicated with the cavity of the bladder at its neck, by an orifice containing phosphatic calculous matter, and also with the rectum by an opening, of the size of a probe, through its anterior wall. The bladder was very vascular.

[To be continued.]

III.—LACERATION OF THE LOWER PART OF THE RIGHT INTERNAL JUGULAR VEIN, CAUSED BY FRACTURED PORTIONS OF CLAVICLE.

Plate VIII.

This case I have thought worthy of being recorded, as it is in itself, I believe, of rare occurrence; and also because it calls to mind very strongly the case of the late Sir Robert Peel, the mode of whose lamentable death, most probably occasioned, at least in great part, by the wounding of a vein by a fractured clavicle (the left), was at the time looked upon as being almost, if not quite, unprecedented. The accident in this case, moreover, took place within a few months of the death of Sir Robert Peel.

Case.—A young man, aged 23, apparently in good health, was standing in Hyde Park, July 1, 1851, under some trees, in a thunderstorm. A rotten bough fell down from above him, and struck him against some hurdles. He died almost immediately afterwards, and was brought into our "dead-house" at St. George's Hospital quite dead. I examined the body, and found the following lesions:—*

There was great swelling and deformity about the lower part of the neck on both sides, and on opening the chest, and removing the sternum, &c., I found that both clavicles were extensively fractured, and that a very large amount of blood was extravasated about the portions of the fractured clavicles, and also beneath the cervical fasciæ. Fracture of the second rib on one side also existed, and in connection with this, there was a large quantity of extravasated blood among the pectoral muscles, and much also in both mediastina. On removing the fractured clavicles, I found that the lower part of the right internal jugular vein, at a point about one quarter of an inch above its junction with the subclavian to form the innominate vein, had been lacerated by depression of some fragments of the broken clavicle. The lacerated opening was oval in shape, its longest diameter being across the vessel, and was of such a size that it

* See St. George's Hospital Path. Museum, series vi, No. 172.

would about have admitted an ordinary sized pea. There was nothing like firm coagulum around or within the injured vein. The jugular vein of the opposite side was uninjured, and the subclavian veins on both sides were unaffected. On further examination some broken ribs were found on one side, and the lung corresponding was lacerated; moreover, the sternum was found to be fractured. Blood was also found extravasated between the peritoneum and the abdominal muscles. Nothing worthy of note was further observed in the body.

Observations. — Death was, no doubt, occasioned by hæmorrhage into the mediastina and the tissues of the neck, and among the muscles, &c. At least this must have contributed very materially to the fatal issue; and it is very clear that even if medical advice had been at hand at the moment of the accident, it must have been unavailing. How far the physical appearances presented by the neck and parts around the fractured clavicles resembled those in the case of Sir R. Peel, above alluded to, is of course entirely a matter of conjecture; but it is quite possible that examination of these parts after the accident, and before death, would have shown, as in Sir R. Peel's case, the existence of a "swelling" pulsating to the touch, and resembling a "diffused false aneurism." Of course the two cases so far differ, in that we have in the one case the lower part of the internal jugular vein lacerated, whereas in the other "it was supposed" that it was the subclavian vein which was injured.*

I do not see how it would be possible by ante-mortem appearances to diagnose laceration of the subclavian, from that of the internal jugular vein, at a point so low down as the injury in the present instance. As in Sir R. Peel's case, fracture of the ribs was found in this one also.

The accompanying illustration, Plate viii, which Dr. Westmacott made at my request, shows well the position, and size, &c., of the lacerated opening in the jugular vein. The proportions between this and the subclavian vein have been unfortunately not preserved, owing to the shrinking of the latter by reason of its not having been distended whilst immersed in spirits of wine.

* It will be remembered that there was no post-mortem examination of Sir R. Peel.

OF THE BRANCHING OF NERVE TRUNKS, AND OF THE SUBDIVISION OF THE INDIVIDUAL FIBRES COMPOSING THEM.

By Lionel S. Beale, M.B., F.R.S.

Plate IX.

Everyone who has endeavoured to follow by dissection the bundles of nerve-fibres which compose the large compound trunks, and trace them as they ramify in the branches, knows that it is not possible to track a particular strand of fibres for any distance, in consequence of the frequent changes which occur in its relation to other fibres entering into the formation of the compound trunk. A bundle of fibres may often be shewn to divide into two smaller bundles. Of these one may, perhaps, help to form a branch, while the other may continue onwards in the original trunk. Bundles of nerve-fibres often cross from one side of the trunk to the opposite, and very frequently, even by coarse dissection, a twisted arrangement may be demonstrated. In fact in all cases, although the constituent bundles of a trunk, and the constituent fibres of a bundle, appear to run parallel to one another, they really interlace very freely and cross one another frequently. It is therefore impossible to divide any nerve trunk longitudinally without cutting across a great many fibres; indeed it is probable that if a nerve trunk were simply slit for some distance in the longitudinal direction, all its fibres would be cut across as effectually as if the entire trunk had been severed transversely.

The arrangement of the individual nerve-fibres, figured in Plate ix, in the smaller, and even in the smallest trunks, is precisely the same as that of the bundles composing the larger trunks. The arrangement is constant, too, in all animals, and therefore seems to be essential or fundamental. I adduce it as a broad and most important fact in support of other arguments I have advanced in favour of the view that nervous organs invariably form complete circuits, and I hold that a circuit is a fundamental necessity for nervous action.

The arrangement presently to be described is at once explained, if there be outgoing and returning fibres in the same nerve trunk. It cannot be accounted for upon the hypothesis of the fibres being motor and sensitive, or afferent and efferent, in the same trunk, because the arrangement exists and is quite as striking in purely sensitive as it is in mixed nerves.

It is incumbent upon those who maintain that nerves terminate by distinct ends in tissues to offer some explanation of this peculiar arrangement, which seems quite inconsistent with the view they uphold. Neither Kölliker nor Kühne, nor any of the German observers who have lately been writing, seem to have

noticed this remarkable fact with regard to the course of nerve-fibres in trunks, although I have referred to it in my recent papers, and have drawn attention to it in my lectures for three or four years past. Dr. Arnold, of Heidelberg, has indeed lately described the arrangement as seen in the nerves of the Iris. (*Virchow's Archiv.* vol. 27, p. 345, 1863.) But it occurs in all nerves.

My friend Dr. Ciaccio has also delineated the arrangement as it exists in the nerves of the cornea (*Mic. Journal*, July, 1863). It is however not peculiar to the nerves of these textures, but is constant in all tissues. (See my paper "On the Ultimate Distribution of Nerves," *Archives of Medicine*, vol. iii., p. 236, plates xv., xvi., xvii, 1862. Also, *Phil. Trans.* 1860, Plate xxiii., Fig. 8.)

I will now briefly describe the arrangement as it may be observed in the nerves distributed to the pectoral muscle of the frog, and in those which supply the mucous membrane of the palate, the skin, and other parts.

The very first bundles resulting from the subdivision of the large trunk, are composed of fibres, some of which come from the nerve, while others seem to return to the nerve, or in other words the branches from the trunk contain fibres, which come from the centre, and fibres which pass towards the centre.

The lines drawn in Figs. 1 & 2, Plate ix, may stand for fine nerve-fibres, or bundles of nerve-fibres, for the arrangement exists in fine nerve-fibres, and in bundles composed of finer compound bundles. It is noticed with respect to the roots of the nerves as they pass from the cord. (See the papers of Mr. Lockhart Clarke, *Phil. Trans.*)

It is observed in the case of the fine nerve-fibres ramifying in the brain; it is found in all ganglia; it exists in the skin, in the fibrous tissue of the abdomen, and in the cornea of the frog, and is very easily demonstrated. The nerves distributed to the papillæ of the frog's tongue divide into two bundles at the base, which pass from the papillæ in opposite directions, while at the summit the fibres divide into very fine branches, which are connected with very numerous nuclei, and form a terminal plexus or network, which is situated at the summit of the papillæ, Plate ix, Fig. 5.

Fig. 3, Plate ix, is taken from a preparation of the pectoral muscle of the frog. It illustrates the manner in which nerves divide in the trunks, and the course taken by different fibres. Each fibre resulting from the division passes towards its distribution in different branches of the nerve.

Fig. 6 is also from the breast muscle of the frog, and represents two branches coming off from one trunk. Observe the

fibres dividing at *a*, *b*. The arrangement is shown more distinctly in Fig. 2, and I have placed arrows in the direction in which I conceive the nerve current to be passing. The arrangement is such that the currents pass in opposite directions in the fibres which are contiguous.

Fig. 4 is from the mylohoid of the hyla, and represents some of the finest nerve-fibres visible. It is magnified 1800 diameters.

It has been shown that a crossing of the nerve-fibres in trunks occurs, that the passage of nerves from the same bundle in opposite directions is constant in all tissues, and in all animals, and it has been conclusively proved, at least in many tissues, that such an arrangement is associated with the existence of a most intricate terminal network or plexus, composed of very minute fibres. Is it not therefore almost certain that in muscle and in all tissues a similar terminal network or plexus exists? If not, how is the peculiarity to be explained? If the nerves terminated in free extremities they would surely pass to their destination by the shortest route. The argument seems conclusive even in the absence of all demonstration. No one would be so rash as to assume that such an arrangement as that figured is purposeless and accidental. From its constancy it is most certain that it is essential, and from the fact that it exists in the case of old fibres, fully formed fibres, and in the youngest fibres, it is certain that the arrangement is necessary to the very nature of nerve distribution.

The facts described in this paper give support to my view with regard to the existence of nerve circuits—a view which is fully justified by the evidence given in former papers. How the arrangement is brought about shall be fully discussed in another communication. My paper, on the structure and formation of the so-called a-polar, uni-polar, and bi-polar nerve-cells of the frog, (*Phil. Trans.* 1863,) which will be published in a month, contains numerous observations which strongly support this conclusion, but from another point of view.

The general inference I deduce from a careful examination of the course taken by nerve-fibres in the largest and smallest nerve trunks, in bundles of nerves ramifying in nerve-centres and in peripheral parts is this:—That with regard to every nerve-cell, central and peripheral, there are—

1. Fibres, or at least a fibre, passing to the cell.
2. Fibres, or at least a fibre, passing from the cell.
3. Commissural fibres, passing between different cells and connecting with each other certain cells and sets of cells, which concur in their action.

In Fig. 2, Plate ix, *a* and *d* may be commissural fibres, *b* may be a fibre passing from, and *c* a fibre passing towards a central nerve-cell.

RECENT CONTRIBUTIONS TO THE STUDY OF CONTINUED FEVER.

No. I.

At the last gathering of the Social Science Association at Edinburgh, under the Presidency of Lord Brougham, Professor Christison delivered a very important address on Public Health, in which he discussed all the views advanced at different times upon the operation of external agencies in the production and spread of diseases of zymotic type, and expressed his opinion as to how far we might hope to prevent such diseases, or limit their spread, by the removal of such causes as dirt, defective drainage, bad air, overcrowding, &c.

An address, on such a subject from so philosophical a thinker, and so experienced a physician as Dr. Christison, naturally attracted a large amount of attention and discussion.

As our readers have doubtless already read that address elsewhere, we shall assume that they are conversant with the facts and reasoning advanced by Dr. Christison, but we have thought it well to call attention to it by way of preface to the papers analysed in this article.

En passant we may remark, however, that Dr. Christison's opinions seem to have undergone some modification since 1846, for, in the July number of the *Edinburgh Medical Journal* of that year, is a paper by him on "Typhoid Fever apparently originating in local miasma." The epidemic, he there describes, was so limited in its sphere of action, and virulent within the circle of its influence as to have given rise to suspicions of poisoning, although it really depended upon the disturbance of a soil which had been for a long time impregnated with the products of sewage.

The statistical method of investigation applied to questions of general health is open to many fallacies, among which may be adduced the stringing together under the category of "Typhus" all the continued fevers of this country, and, similarly, under the single term of "foul air" comprehending all kinds of foul air, produced by many and different agents.

Into the etiology of true typhus we do not propose to enter specially, further than to remark, that, in its prevalence, it has partaken more of an epidemic than an endemic character; that it proves eminently contagious when it has originated, is allowed on all hands; that there are good grounds for the belief that it can be generated by the overcrowding of human beings, debilitated and enervated by starvation, during the prevalence

of cold weather, but that its spontaneous origin (and the same may be said of typhoid fever) cannot yet be considered a settled point.*

From the last Army Medical Blue Book for 1861, just published, we have extracted the following observations from the various papers on fever.

Professor Christison holds the theory that diseases change their type, and that the generation and spread of these have some reference to epidemic constitutions of the season prevailing at different times.

Deputy Inspector-General Lawson's paper is upon the influence of "Pandemic causes" in the production of fever. In elucidating this subject he has laid under contribution the Army Statistical Returns of Sickness and Mortality, ranging, in time, from 1817 to 1856, and, in space, extending from Ceylon to Jamaica in longitude, and from Great Britain to the Cape of Good Hope in latitude.

Dr. Marston, in his report on fever, says:—

"It is highly probable that some atmospheric conditions 'beyond our ken' enter into the creation and spread of the fever poison. If within the limited area presented by a regiment or garrison we can more leisurely and exactly trace the steps of progression of a disorder, it must not be lost sight of that there are other causes and influences at work, upon a far larger scale—waves of diffused influence."

This, then, is the subject of Mr. Lawson's paper, and, from an examination of the data afforded by the official returns, he is enabled to trace the curious regularity "and progression of the oscillations of febrile disease at so many points on the earth's surface, far removed from each other, and embracing the continued, remittent, and yellow forms of the disease, as to leave no doubt of the operation of some cause equally extensively." . .

"The facts detailed establish conclusively the occurrence, between 1817 and 1836,† of a series of oscillations of febrile disease, following each other with amazing regularity. These have been attributed to a cause or influence which, from its extent and progressive character, has been designated a

* In the case of the "Schah Gehald," wherein it was asserted that "typhus" had been spontaneously generated among its living cargo, under circumstances parallel with those stated in the text, this has not been quite substantiated, because there was an absence of proof that none of the passengers or crew were affected at the date of their first embarkation.

† At the end of this paper the reader will find such a brief summary of the facts adduced by Deputy Inspector-General Lawson as will serve to indicate the manner in which he has worked out his subject.

'pandemic wave,' the term epidemic referring rather to a single form of disease affecting a limited space." . . .

"What the nature of this pandemic wave and its mode of action may be, it is difficult to say; but certain deductions may be made with advantage from the facts concerning it set forth above. Of these the most striking is the constant course in the Atlantic and western part of the Indian Ocean, from south or south-east to north or north-west. This can take place in virtue of some law of nature only, and the situation of the magnetic poles immediately suggests itself as being in some way connected with it; but of this there is not at present any proof so far as I am aware." . . .

"Another point of more immediate importance, as bearing on some of the medical opinions of the day, has been alluded to several times in the course of this paper, viz., the occurrence of every form of fever under the operation of the same pandemic wave, yellow fever, remittent, intermittent, and continued fever, and plague, having prevailed in the localities in which they were endemic as the wave passed over them."

"As it appears that in different countries different forms of fever prevailed, under the same general influence, it must be admitted that the pandemic cause determines the frequency and severity, rather than the particular form of the fever, which, there are many reasons to conclude, is more intimately connected with the local circumstances at the time."

"Sometimes, it is true, one of these forms invaded the territory of another for a while, and such intrusions have given rise to some of the most embittered controversies in the history of medicine. The principles here developed will place the evidence in connection with these unusual irruptions of disease in a new light, and contribute to reconcile the opposite opinions regarding them, giving us a basis to reason on epidemics better suited to the time, and the advance of our knowledge of the causes of disease, than that on which the subject was contested by Pym and Bancroft fifty years ago." . . .

"It is an interesting subject of inquiry whether the pandemic cause acts on the system, rendering that more susceptible of the influence of the ordinary causes of disease, or on these themselves, giving origin to a more concentrated, or a different kind of poison, which generates a more virulent, or a new form of disease; or whether it affect both, increasing the activity, or altering the kind of the causes, and enhancing the liability of the body to be affected by them. Our information on these points is still very limited and unsatisfactory. I have observed that prior to, and during the prevalence of, an epidemic of

yellow fever, the urine contained an unusual quantity of urrhodine, giving a deep reddish-brown when treated with nitric acid and set aside for some hours. Something similar has been observed with regard to cholera. These tend to show that the body itself is affected by the pandemic cause; but the observations require to be repeated, before much weight can be attached to them."

"The outbreaks of epidemic disease have been referred immediately by some to the rapid generation and diffusion of myriads of miscroscopic animals, and by others to a similar production of vegetable organisms. The evidence in support of either is very defective. It may be well to keep the possibility of such things occurring in view, while we cannot be too cautious in admitting their efficiency, without adequate and irrefragable proof."

"In the above remarks, the influence of the pandemic wave in producing fever only has been examined, but it is obvious in many other affections, of which cholera, diarrhœa, dysentery, and scurvy are the most important. Even consumption and ulcers vary so much from time to time in frequency, as to suggest that they are similarly affected."

Mr. Hanbury, Surgeon to the 33rd Regiment, describes his experience of typhoid fever in India, from a sanitary point of view.

The clinical and anatomical characters of the disease, illustrated in the cases he details, were, in his experience, of an unusual description. He indicates how this particular form of fever might easily have been classed under the head of the remittent fever of India, occurring, as the cases did, during the autumn months of the year, at a time when cases of intermittent and remittent types of fever were to be expected, and were actually present, and where the symptoms presented by many of them were suggestive of those diseases rather than of typhoid fever, which the post-mortem evidence proved them to have been. He traces the rise, prevalence, and diappearance of the disease in connection and relation with local sanitary causes.

In 1859 the garrison of Malta numbered 6,744 men, and 1,196 cases of fever (of all types) were treated, with a mortality of 41. Dr. Marston takes this as his text, upon which he relates his whole experience and observation of this class of disease.

The fevers considered are:—1. Typhoid; 2. Gastric—Remittent, and Bilious Fevers; 3. The three to five day fever of the Mediterranean.

Typhoid fever was the cause of the mortality in the garrison of Malta in 1859, and with this he commences. He considers the question of the non-identity of the typhus and typhoid types to be settled. He traces the introduction of typhus fever into Malta upon two occasions,—during the Crimean war, and in the spring of 1861, by the crew of the Egyptian vessel "Schah Gehald," which had attained such a morbid notoriety at Liverpool. The disease proved eminently contagious, but spread within an extremely limited range at Malta. He discusses, at some length, the etiology of typhoid fever, and its relations to other miasmatic diseases. He endeavours to prove that Dr. William Budd's and Professor von Geitl's theory, as to the spread of this disease by contagion only, and particularly through the infectious properties of the excreta of affected individuals, cannot include *all* the facts.*

Dr. Marston expresses some hesitation in the acceptance of these doctrines.

"We are in a condition to say that no one has hitherto produced any organism without the agency of pre-existing germs, and, conversely, that wherever such production has been the subject of observation and experiment, the conditions permitting the presence of such germs have never been absent; but it is to beg the point at issue, to say that the generation of typhoid fever rests upon the same basis as this, and that the impossibility of tracing the lineage is of no more force against propagation by continuous succession in the one case than the other. For contagionists to assert that what is not impossible may have taken place, is a secure way of guarding their theory, which would be only justifiable if there were the enormous preponderance of facts upon their side, such as we possess with regard to the transmitted descent of animals and vegetables. The numerous occurrences will remain unexplained, wherein intestinal fever has suddenly appeared in a limited and previously healthy locality, concurrently with a foul state of sewage, &c.—the progress of the disease ceasing with the removal of this, and the most careful inquiries having failed to trace the arrival or presence of an infected person from without

* As is well known, Dr. Wm. Budd has, in some very able papers recently published, not only endeavoured to establish the general contagious nature of this disease, but to fix the contagious and infectious properties upon the excreta from affected individuals, classing typhoid, cholera, and yellow fever in the same category, viz., ground infecting poisons. Dr. Budd is a total disbeliever in the spontaneous origin of this intestinal fever. He says that, to assert this upon the merely negative evidence of our inability to trace the continuity of the succession in this instance, we might, equally, hold the spontaneous generation of animals and plants.

—if the occasional spontaneous origin of the disease be not received."

In tracing the origin and course of the epidemic in Malta, Dr. M. appears to take the following grounds:—

1st. Some wave of diffused influence, or unknown atmospheric conditions which enter into the creation and spread of the fever poison.

2nd. A concurrence of local causes, such as impure drainage and sewage, giving use to fœcal odours and the products of animal decomposition, at a time coincident with such barometrical and thermometrical states of the air as fit it for the reception, absorption, and subsequent deposition of miasmata and malaria.

3rd. Contagion, whether arising from intercourse with affected individuals or exposure to excretions from their bodies.

That these influences may vary in the importance of the position they occupy, as causes of the spread of the disease in different times and places, is very probable. In the Malta epidemic the local causes seemed to hold the most prominent, and the personal causes a subordinate position, in this respect. In a small village this may be reversed, and contagion may come to occupy the first place. There can be little doubt that fever may spread in more than one way, and there is no more curious or important study than the varying combination of the epidemic, endemic, and contagious influences. Nay, further, the modification of fever (any form) by local causes is well shown by what Sir James McGregor says of plague, in the different localities of Egypt—sometimes taking on malarious, at other times the inflammatory type, &c.

From the facts detailed, he shews, that the fever appeared in one new barrack shortly after the opening of some sewers, and that, generally, the amount of the disease, and its severity, had an intimate connection with the inferiority (in a sanitary point of view) of the barracks occupied. And, on the other hand, he points out that in some forts, remote from barrack and town sewage, the disease was for a long time absent, and was very limited when it did appear.

He sums up the result of his observations on the causes of enteric fever in the following propositions:—

1. That typhoid fever is endemic rather than epidemic; when the latter, it is often remarkably circumscribed within one or more limited spheres.

2. That it is, in all probability, contagious; but that this is inadequate to explain all its outbreaks, and that contagion is to

be held in the light of a subordinate rather than a primary and principal cause of its spread.

3. That typhoid fever is, probably, etiologically allied to other diseases, such as gastro-enteric ones, having either a different or allied patheology.

4. That, in its origin and spread, it has an intimate connection with bad drainage and fœcal fermentation.

5. That the products of such fermentation may be varied in many ways, and that so, in all probability, they may also give rise to diseases differing from typhoid fever.

6. That enteric or typhoid fever is probably capable of a spontaneous origin, as opposed to that of continuous succession from the bodies of individuals under its influence.

7. That it ordinarily commences about autumn, and reaches its acme of prevalence soon after that season, continuing through the winter in mild climates, but ceasing towards the spring.

8. It is probable that the character and constitution of the season exert some influence. Qy. It has appeared in Malta more than once, after hot and dry summers, with the first autumnal rains.

He then analyses the different symptoms presented by the patients during the course of the disease.

Upon the subject of exanthem, he contrasts the differences between the typhus and typhoid eruptions, and adduces some cases in which purpuric or petechial spots have appeared with the "rose" papules, characteristic of typhoid fever; but such petechial spots have not pursued the gradual and continuous course of the typhus eruption, and are, he thinks, only to be considered as accidental to the stagnation of the blood in the capillaries, the impaired contractility of the skin, and the asthenic condition of the patient. In such cases the eruption, as a whole, consisted of the characteristic effaceable "rose" papules of typhoid fever, which came out and passed off in successive crops.

Vesicles sometimes, though excessively rarely, appear upon the summits of the spots. The medical officers were in the habit of testing any doubtful spot, which might be found in a case where the diagnosis was uncertain, by noting its character, duration, and whether it vesicated or not; if it did so, this was deemed, as far as it went of course, as negativing the diagnosis of typhoid fever. Two exceptional cases occurring within his own observation led him, however, to modify that opinion.

The presence of the rash seemed to import little, if anything, for it occurred in the mild as frequently as in the severe

forms of the disorder; and the cases attended with a copious rash were not at all more severe than others.

Under his remarks upon the pyrexial character of the symptoms he alludes to cases simulating a masked intermittent fever.

Headache and delirium do not appear to co-exist, nor was the latter an early symptom in any, except the worst cases, where the amount of urinary excretion was very markedly diminished. As the headache passed off, delirium—if it appeared at all—set in. Sleeplessness, dreams, and a vague sense of headache were among the earliest symptoms.

Jaundice—which is very rare in typhoid fever—was present in three cases, and in one of these, the remittent type of the symptoms during life led to the impression that the patient was suffering from a form of "bilious remittent fever."

In reviewing the symptoms referable to the digestive system he remarks, that tolerance of pressure of the abdomen was out of all proportion to the amount of anatomical lesion present in most of the fatal cases.

Omitting his observations upon the diarrhœa, the characters of the stools, and the symptoms referable to the abdomen of the typhoid fever patient, we pass to—

Hemorrhage from the bowels. Of five cases in which this was observed, four died. Great differences of opinion seem to exist as to the importance of this symptom. Inasmuch as it may arise from different sources and causes, so must the gravity of its import vary. As it exudes from an abraded fungous granulating substance—from arterial or venous implication in the ulcerating or sloughing process—so will the occurrence of hemorrhage vary in amount and danger.

Intestinal hemorrhages, from insidious ulceration, are not uncommon during apparent convalescence, and serve to indicate the great care required in diet during such times.

Under the head of *renal functions* we find nothing new. He says—the worst cases,—of an adynamic type,—cases apparently labouring under the directly prostrating effects of the disordered blood upon the nerve centres—excreted urine remarkable for the diminished amount of water and solids. In one case, which was complicated with icterus, the patient excreted only 7 oz. of urine (specific gravity 1030) in forty-eight hours, and this was albuminous, and loaded with bile pigment. He died apparently of coma, the result of uræmia. In no case of pulmonary disease was there an entire absence of chlorides. Patients (during the later stages particularly) complained of urethral scalding and difficulty in voiding their urine;

but retention of the urine, from functional paralysis of the bladder, only occurred once.

Dr. M., in speaking of the occasional occurrence of dropsy of one or other of the lower extremities, remarks, that it probably depends upon one of two causes, viz., the entrance of something into the veins from the intestinal ulcers, giving rise to embolism, or upon a spontaneous coagulation of the blood within a vein.

In his remarks upon the duration, and varieties, in point of fatality, of this fever, he says:—The cases, that were at all severe, rarely improved under three weeks or thirty days. There is great difficulty in arriving at an accurate date as to the termination of the fever. The result of clinical and post-mortem observation would lead to the inference that the typhous material commences to soften about the end of the second week, and that the ulceration and elimination of this deposit proceeds energetically, and, as it were, critically during the third week. In moderately severe cases by about the thirtieth day, the typhous material is eliminated; but the ulcers remain soft, denuded, and in various states as to vascularity for a much longer period. If the ulcerating process set up in the intestine continue, as it often does unfortunately, this is, of itself, sufficient to cause the phenomena of the fever and, in this way, a case may be prolonged until the sixth week or more, and terminate ultimately by perforation of the bowel.

In the diagnosis of typhoid fever he conceives that the chief difficulties will arise from the absence of the exanthem, or from the limited period of our observation of the patient. The first (exanthem) renders the diagnosis certain; but in the absence, even, of this we may generally arrive at an accurate conclusion by attention to the symptoms. The two diseases most resembling typhoid fever are acute tuberculosis and certain cases of pyæmia.

Dr. M. then enters into the consideration of the pathology and morbid anatomy of the disease. His observations are in accordance with the descriptions usually given.

He arranges the condition of the intestinal and mesenteric glands under two groups.

1. A deposit of typhous material in the glands, variable, but large in amount, with great local congestion or active inflammation of the mucous membrane in their neighbourhood.

2. A minor amount of deposit and swelling of the glands with, relatively, little or no inflammatory action.

When the deposit is very large, it can be seen to slough away; when less in amount, but still large, portions of the new and germinating formation, remotely placed for vascular supply, will

die in small pieces; while another portion will undergo a still finer and molecular necrosis, and the remainder, degenerating, may be absorbed or pass off with the discharge from the ulcer. So we pass to a condition in which there is a minimum amount of this morbid deposit. Judging from microscopical examination of this, it would appear to undergo a process, which Virchow terms "tuberculization," becoming a fatty, granular, and pigmental mass, and, as such, to be absorbed either without any, or with a very trifling loss of substance in the mucous membrane.

He thinks that the mildness of the general symptoms and shortness of the course of the fever, in certain cases, render it highly probable that the disorder is occasionally aborted, the amount of deposit being small enough to be removed by absorption. The fact that the tumefaction and deposit in the mesenteric glands must be so removed is an evidence that a similar process may occur in the intestinal glands.

While the different morbid states of different intestinal glands, in regard to the active local processes going on in one or more of them, may be a cause of the fluctuations observed in the patient's febrile condition from day to day, he does not think that this can be the sole cause, because such changes were observed in a few cases that proved fatal before any softening or ulceration of the deposit had taken place.

He conceives that it is to the admixture of certain products from degenerating and ulcerating typhous material with the blood, that many of the inconstant and irregular pyrexial states and local complications, occurring at a late stage of the disorder, may be due.

Of seven instances of peritonitis from perforation of the gut, he mentions one in which the perforation of bowel took place from without by the opening of an abscess in a mesenteric gland into the interior of the intestine.

Passing over his observations upon the morbid changes in the spleen, lungs, &c., we notice that he mentions three instances in which he observed the products of inflammation within the pericardium.

Under the head of air in the venous system, he describes three cases in which this existed. In all three the death was rather sudden.

The gas was not the product of post-mortem decomposition in the above cases. There were certain conditions common to them all, viz., the blood was dark and fluid throughout the body, and bubbled out from the divided veins. In the first two cases, there was an emphysematous state of the tissues of the

neck and chest, besides air in the right side of the heart, hepatic and portal venous systems, venæ cavæ, &c. The lungs were emphysematous, but no air was expelled by inflating them. In the third case, the air was only present in the abdominal veins (portal system). In this instance, the patient had lost a large amount of blood by stool and, although the lesion of the vein was not discovered, it is probable that it may have entered a vessel, through an ulcer in the bowel.

There were not any ulcers in the larynx in the above cases. Dr. G. O. Cless, in his memoir upon this subject, relates similar instances, all cases of typhoid fever, and is of opinion that the air is generated during life by a spontaneous decomposition of some of the constituents of the blood. Dr. Wilks has shewn that emphysema may result from the air entering through ulcers in the larynx. During the Crimean war, it will be remembered, that similar facts were recorded by different observers.

The remainder of this division of the paper is taken up with a description of cases which died during apparent convalescence, or were the subjects of a relapse of the fever with the exanthem.

The writer conceives that atrophy of the coats of the intestine and very grave impairment of the nutrition of the body may succeed this fever in some cases, of which he relates instances.

[Our want of space prevents our entering into the remaining portions of the paper.]

Appendix Referred to at Page 3.

Mr. Lawson—after giving a table of millesimal ratios of deaths from intermittent, remittent, yellow, and continued fevers among our troops during each year, from 1817 to 1836, at all the foreign stations—furnishes us with tables of the admissions from continued fevers at the Fever Hospitals of London, Cork, and Dublin, and the Royal Infirmaries of Glasgow and Edinburgh.

He proceeds to show the striking evidence of the passage of the pandemic wave derived from the outbreaks of disease in various civil populations.

"Thus petechial typhus was rife at Naples in 1817, and a disease denominated malignant typhus prevailed at Algiers the same year. In 1818–19 plague ravaged Morocco and the northern coast of Africa, and in 1819–20 it prevailed at Constantinople. Yellow fever prevailed in the south of Spain with

varying intensity from 1819 to 1821. There was great mortality from typhus in Ireland in 1817–18–19, and again in 1822. Fever was also very common both in England and Scotland during the former period."

"The pandemic wave, which was still sensible in the Ionian Islands and Canada in 1817, overspread the British Isles in 1817–18–19; and it will be observed that London and Cork were, comparatively speaking, affected to a greater extent in 1817 than Dublin, Glasgow, or Edinburgh; all except London had their greatest sickness in 1818, but the Scotch stations carried more of this into the following year than the Irish. The wave described previously as having passed over the Mediterranean in 1818–19 was experienced much more slightly; it seems to have kept up the admissions in London during 1819–20; in Dublin, in 1820–21; and in Edinburgh till 1822. The wave which was at the Ionian Islands in 1821 was sensible in London and Cork in 1822, and in Dublin and Glasgow in 1823. This was followed by that which was at the Ionian Islands in 1823, so closely, that fevers increased in numbers for several years, as will be shown below."

Again, between 1823 and 1829, there was a series of pandemic waves, first apparent at the Cape, from which they passed on to the northward in succession with remarkable regularity.

"In 1824 the first Burmese war commenced, and troops were sent to various points of that territory, who suffered much from fever in this and the following year. Those at Rangoon were affected early in 1824 with the 'Duegur,' or what has been called the 'Dandy fever.' This form of the disease appeared at Calcutta at the same time; the following year it appeared at Burhampore and places in the vicinity. Plague broke out at Cairo in 1824, and carried off 30,000 persons, the following year it prevailed in Lower Egypt. It was rife at Constantinople in 1826, and in 1827 reached Odessa. In 1828-29 plague was very common in Egypt, Syria, Greece, and Turkey in Europe, and affected the Russian troops in the Danubian Provinces, and in the neighbourhood of Adrianople extensively. Petechial fever prevailed at Naples in 1828. In 1827 remittents of a very aggravated type were common in the fenny districts in the south of England. In Scotland, fever was common in several of these years."

"It was stated above that the wave embracing the Ionian Islands in 1821 was sensible at London and Cork in 1822, and at Dublin and Glasgow in 1823, and that this was followed by that found at the Ionian Islands in 1823–4, so closely that sick-

ness increased for several years, attaining its maximum in London and Ireland in 1826–27, but in Scotland, as before, a year later, when the stations to the southward were again healthier. In Iceland, typhus, with some other forms of epidemic disease, prevailed in 1828–29, a year later again than in Scotland. Remittents, equalling those of the tropics in violence and unmanageableness, were common in the marshy districts in the south of England in 1827."

THE NERVES OF THE ORGANIC MUSCLES.

By Dr. Klebs, Assistant at the Pathological Institution at Berlin.

The bladder of the frog has served exclusively as the object of my researches. As the lateral parts of the bladder and the ligaments contain the larger nerves and vessels, this organ appeared particularly adapted for the discussion of the question, "In what manner are the nerves distributed to the tissue of the organic muscular fibres?"

The more important results of these researches may be stated as follows:—

1. The nerves approach the hindmost part of the frog's bladder from both sides of the rectum in several branches, anastomosing amongst each other. A main branch rising to the top of the bladder is perceptible on both sides. From this plexus, which does not extend over the foremost part of the bladder, run in all directions, a great number of smaller branches, at right angles to the ascending fibres, which divide again and again, until they only consist of one or two fibres. This formation of the branches only takes place in a very limited space, hardly extending over the third part of the posterior surface of the bladder in its full height, and growing narrower towards the top. It is but in the same limits that ganglion cells are to be found. The larger the branches of nerves in which they are included, the greater is the number of cells forming one ganglion. On the outlines of that place only a few single cellules are perceptible, either attached to one of the smaller branches, or connected with a single nerve fibre, "final cells" (enständige zellen) according to Jacubowitsch.

2. The larger branches consist throughout of double contoured fibres, the ganglion cells being precisely similar to those of the sympathetic. The connexion existing between the two can only be ascertained on the peripheral branches of the nerves.

3. The nerve fibres of the larger branches are enclosed in a tubular neurilemma, showing, under the microscope, on both sides of the fibre two lines, between which longitudinal nuclei will be perceived. The marrow-like (myeloid) substance of the fibres is separated from the neurilemma by a small transparent space, and the former in its fresh state is bounded by two stripes of a bright hue, in the middle part of which the nerve fibre assumes a transparent appearance. Counting the neurilemma as a part of the fibre, one may call it "an eight-lined one."

In the further progress of the nerve fibre, the neurilemma is getting narrower, only one line is perceptible, and the nuclei when seen from the side view are generally situated so that the pointed ends seem to extend into the visible contour of the neurilemma. On still advancing, the neurilemma joins the nerve-substance, the transparent space ceasing, and the nuclei seeming to be laid on the marrow-like substance. Meanwhile, also, the nerve-fibre has altered its qualities; only very little narrowed, it appears throughout of a plain grey colour, the bright stripes disappear more and more. Then generally follows a portion, in which the nerve-fibre affords the view of fine longitudinal stripes, an alteration which takes place rather suddenly, particularly at the division of the fibres. These latter fibres (or bundles of fine fibres) dissolve by detaching small pale fibres, which equally carrying nuclei anastomose amongst each other, and form a wide-meshed network ("net-fibres, intermediate network") showing in the knot-points for the most part a triangular accumulation of a fine granulated mass, which, however, must not be considered as ganglion-cells, because nuclei are not to be found at all places. The network of the nerves appears to be completely closed, in which formation, participate all the nerve-fibres leading to the bladder. Within that network sometimes larger accumulations of fibres make their appearance, from which net-forming fibres branch off in all directions. These masses of fibres seem to possess a different development in each individual case. In some instances I did not observe them at all. Dr. Beale, in his last publication on that subject, gives a sketch of them developed upon an extensive scale (Archiv. of Med. vol. iii, Pl. xv.).

The fibres included in these masses, are much finer than those which form the network; I estimate them at about 0,0001 mm., whilst the net-fibres are of a size far as 0,0003 mm. They are principally corresponding with the fibres of the long-striped nerves above alluded to, and this leads me to believe, that all the pale nervous fibres lying between the two consist of bundles of the

finest fibres. From the same masses sometimes radiate several fibres enveloped in the same neurilemma, which are separated by wide intervals. The nuclei were placed partly in the neurilemma, partly in the single fibres themselves. In the latter place they appeared a little more pointed, I cannot assert whether or not these compound fibres, which are similar to those described by Remak and Haerkel in the abdominal marrow of the Astacus fluviat., become transformed into pale net-fibres, by the conjunction of their fine fibres, and increasing narrowness of the neurilemma.

4. From the network-fibres the muscle-nerves branch off; some of them originate from the striped, but never from the double-lined fibres Single fibres may be seen parting from the network-fibres in a straight direction, and partly of considerable length, which never enter into contact with each other. They are going to a bundle of muscle-fibres, entering therein under a right or a little more acute angle. Shortly before entering, they show constantly a swelling of a particular kind, which distinguishes itself remarkably from the nuclei by a bright, reflecting appearance. Their form, also, is very characteristic, showing the peripheral end finely pointed, the central one, however circular, often swollen to a sort of ball. In several instances I observed in them reflecting masses, which I may call nervous knots (Nervenknoten), a round spot of a somewhat darker colour with a distinct border-line. The length of these knots exceeds that of the nuclei, and was in one case 0,0083 mm.; the greatest width was 0,0017 mm.; the thickness of the nervous-fibre 0,0003 mm.

The fibre coming out of the knot enters immediately into the bundle of muscle-fibres, in most cases at right-angles to it; divides then into two fibres, separating towards the muscle-fibres, in opposite directions. These latter, "intermuscular fibres," now form long-meshed nets, by splitting themselves dichotomously in such a manner, that the branches, parting at the place of their division, more or less rectangular, turn round again into the longitudinal direction. Each mesh of the net so formed is stretched out in the direction of the muscular fibres, but very contracted in the opposite one. The smallest meshes correspond in their width with that of one muscle-fibre, whereas they never reach their length. All nerve-fibres are going on in a wavy line, and thereby, and by their brighter appearance, they may be distinguished from the pale outlines of the muscular-fibres. The more successfully a preparation is made, the more closed will the nets be seen, whilst in preparations of an inferior kind, many of the fine nervous fibres become varicose, or even disap-

pear, dissolving themselves at last into a line of separated points. I am, therefore of the opinion, that an immediate conjunction between the nervous and the muscular substance does not exist. In the same manner, there is in the voluntary muscles a distinct partition between the two substances, notwithstanding they are in a close contact, a circumstance which may have led to that erroneous supposition, of there being a separating membrane between the two. (W. Krause.)

5. The ganglion-cells of the bladder are, as it is said above, of the same constitution as those of the sympathetic ganglia; somewhat extended cellules with a little heap of red-coloured matter; only in the peripheral parts are occasionally perceptible cells, of a smaller size, and without coloured matter. To each cell runs one double-lined or pale fibre, and from each cell parts also one of the same kind or a small number (three or four) of net-fibres (the latter are the so-called "final cells"). Therefore we have bi- and multipolar cells, but no uni-, and apolar, which is also in accordance with the observations of Dr. Beale (on the structure and formation of the so-called apolar, unipolar, and bipolar nerve cells of the frog. Phil. Trans., May, 1863; Phil. Trans., June, 1862, p. 895; Archives of Medicine, 1862).

6. The nervous fibres exhibit another relation besides that to the muscular fibres and the ganglion cells, namely to the corpuscles of the connective tissue (Bendegewebskörperchen.) To the latter go likewise small fibres which spring out from the neurilemma, which appear very often varicose; but I do not consider this particular quality as a sufficient criterion for their nervous nature, because the same quality may be found in all the fibres diverging from the corpuscles of the connective tissue, and what speaks the more against it is, that in the double-lined nerves, which have a transparent interval between the long-striped or marrow-like ventral mass, no alteration in the inner border-line could be perceived in such places. However, I by no means deny the existence of a connexion between the two structures, which, when judging from the results of physiological experiments, seems rather probable. The only decisive criterion for nervous substance is in my opinion its negative effect as to the longitudinal axis against the colours of plates of gypsum in polarised light, which is by far stronger than the positive one of the diodiaklasts of the muscle-fibre. I reserve a more particular report on this matter, and may here only mention, that this effect may be pursued much further in the peripherical extension of the nerves, than the so-called marrow-like appearance. So all the fibres which form the intermediate net, or

lead to the muscles with their respective knots, will show these particular qualities, whilst hitherto I did not succeed to prove the same state of things on the smaller intermuscular fibres.

I therefore distinguish in the nervous system of the frog's bladder, in the first place a nervous plexus, containing ganglia; in the second one, an intermediate set of nerves, parting from the first, the fibres of it probably representing bundles of fibres; and in the third place, a real intermuscular, probably terminal net, which originates from particular fibres of the intermediate net, furnished with nervous knots.

[Although the author requested me to modify his expressions to suit our language, I have only thought it necessary to alter a very few words, as I felt that by doing more, I should probably impair the author's meaning, which is very clearly expressed. It will be seen, that in the main points Dr. Klebs agrees with me, and especially as to the existence of the network, and the complete absence of any ends or terminal extremities to the nerves. He appears not to have observed the fine nerve-fibres running in the sheath of the nerves, although he has noticed what is generally regarded as the outline of the sheath on one side only of the nerve-fibre (see my Fig. 31, Plate xliv, Phil. Trans. 1862, repeated in Plate xiii, fig. 1, under *d*, in this Journal). Dr. Klebs thinks that the nerve fibres may be continuous with the connective tissue corpuscles. The results of my own observations are strongly opposed to such a view. I feel confident that there is no such connection. Dr. Ciaccio has also expressed himself against such a doctrine (see his paper on the nerves of the cornea. Trans. Mic. Society, July, 1863).—Editor.]

PATHOLOGICAL OBSERVATIONS IN A CASE OF EPILEPSY ACCOMPANIED BY DIABETES.

By J. Lockhart Clarke, F.R.S., &c.

Plate X.

This case occurred in the practice of Dr. Brown Séquard, at the Hospital for the Epileptic and Paralysed. The following particulars relating to the patient's history were supplied to me by Dr. Hughlings Jackson, assistant-physician to the hospital:—

"George M., aged 53, was admitted for epileptiform seizures, April 2, 1862. He was a well-built man, and had had

good general health until his first seizure in October, 1859. Yet he had been an intemperate man, and had had slight symptoms of delirium-tremens, but no distinct attack. There was no hereditary tendency to any particular disease. The only circumstance likely to have had an influence on his illness was that he had had two blows on the head, respectively twelve and five months before the first attack, but they were not attended by any apparent immediate effects.

"As stated, the first attack was in October, 1859. He then had 'fits' for a whole day, and for one year afterwards, he had about one daily. Then he had them oftener, two or three a day. On October 2, 1861, he had fits for the whole day, and from this time he had the fits more frequently than ever, five or six a day, and as many in the night.

"In the severe fits, viz., October, 1859 and October, 1861, he was insensible, but did not bite his tongue. They would begin by a sensation 'in the chest below the left breast, and he was in terrible pain in the stomach.' He was, it was said, first red and then deadly pale, but he did not fall, and was not insensible. He had also attacks in which his limbs were jerked out.

"Careful inquiries were made as to paralysis and defects of the spinal senses, but the only abnormal condition of the kind was that he had incontinence of urine. Of this he passed four or five quarts daily. He drank a great deal and was very ravenous for food. On July 3, his urine was examined for sugar. It was then of the specific gravity of 1037, and contained a good deal of sugar. It was frequently examined and always with pretty much the same result.

"He was subject to headache, and his temper was soured. His mind and memory generally were impaired. He slept very badly indeed.

"He continued much in the same condition until October. On October 13, I saw him at home in consultation with Mr. Powell, of Amwell-street. He had then retention of urine. When I saw him he had been relieved of the symptom, but seemed oppressed and miserable. He still had no paralysis. He had, however, had 'fits' of a different kind. 'He was shaken all over' every two minutes, for an hour. It was not shivering, but we could get no clear account of what took place; indeed, although repeated investigations were made, his wife's statements were very loose, unconnected, and vague. Next day he was incoherent and asked for his mother, who had been dead twenty years.

"On October 29, his fits, according to his wife's descrip-

tion, were as follows:—'His heart first beats violently and he shakes the bed. He turns first red and then white. He is not insensible. They occur about every quarter of an hour.'

"He continued in the same general condition, but towards the end his sight failed, and the left third nerve became impaired, but never completely paralysed. He was now so dull and stupid that it was with difficulty that I could make an ophthalmoscopic examination. I found the optic disc very much reddened, and its margin obscure. In fact the disc was lost in a reddened patch about thrice its size. There were white patches on it in some places. The arteries were very small and the veins large.

"He died April 5th, no other paralysis having been observed. It is certain that, unless at the very last, there could have have been no marked paralysis; but it is possible that a little impairment may have escaped attention, as he was for the last few weeks constantly in bed.

"After his death, I learned that he had had increased sexual desire, and this explained to me curious little scenes which occurred in my presence betwixt him and his wife. He was then so stupid that he did not seem to feel my presence, unless actually addressed."

The above history of the case was furnished me by Dr. Hughlings Jackson, after I had examined the parts which I received for investigation. These parts consisted of the whole of the m e d u l l a o b l o n g a t a and p o n s v a r o l i i, with the crura cerebri and portions of the o p t i c t h a l a m i. The l e f t optic thalamus was almost wholly cut away; but of that on the r i g h t side there remained about the two inner thirds. The right optic thalamus was softer than natural, and its softness was peculiar; for on pressure it returned a certain elastic, or kind of g e l a t i n o u s resistance. On making with a sharp razor a succession of thin sections from without inwards, I found, near the middle of its inner third, a rust-coloured mass about the size of a hazel-nut. This mass, however, was irregular in outline, and extended, in the form of a kind of tapering process, as far as the posterior border of the thalamus, where it joins the crus cerebri. On examining, under a sufficiently-high magnifying-power, different parts of this substance, in a perfectly fresh state, I found them to consist of the following elements:—1. A few partially-disintegrated, or otherwise altered, blood-corpuscles. 2. Hematoidin or colouring matter, resulting from the disintegration of blood-corpuscles. 3. Fatty substance in abundance, and in different forms. 4. Granular corpuscles, known as exudation, or the so-called compound in-

flammation, corpuscles, in considerable numbers. The hematoidin occurred in three different forms. 1. Crystallised in the form of oblique rhombic prisms of different sizes, and either separate, as at *a* (Fig. 1, Pl. X.), or embedded in masses of fat globules, as at *b*. 2. In the form of amorphous granules interspersed through clusters of oil-globules of different sizes, as at *c, d, e, f, g*. 3. In a state of solution in the substance of the oil-globules, staining them with yellow or brown of different shades, as at *b, f, g, h*.

The fatty substance, also, assumed two different forms. 1. Scattered molecules, and globules of different sizes grouped into clusters containing molecular or crystalline hematoidin, by which they were more or less tinted, as at *b, c, d, e, f, g, h, i*. 2. Acicular and radiating crystals of margarin, as at *k*. Granular or so-called exudation corpuscles are represented at *l*.

The rust-coloured mass composed of these elements appears to have resulted either from idiopathic inflammation alone, affecting the part, or from extravasation of blood, followed by inflammation. I am disposed rather to adopt the former explanation. The remaining portions of the thalamus were found to be thickly interspersed with small oil-globules, to which the gelatinous or elastic kind of resistance, already mentioned, must have been due.

The central parts of the pons varolii were somewhat softer than usual, but contained neither fat-substance nor any other kind of morbid product. A similar degree of softening extended down the central parts of the medulla oblongata, involving the middle of the raphè and those portions of the medulla on each side, which are traversed by the roots of the lingual nerves, together with the transverse commissure of the olivary bodies, lying behind the anterior pyramids. The olivary bodies themselves, however, were unaffected.

In the neighbourhood of the calamus scriptorius of the fourth ventricle the posterior parts of the medulla were considerably affected. Fig. 2 represents a transverse section of some of these parts on each side of the median line a little above the point of the calamus scriptorius. *f, e, f'*, is the median portion of the floor of the fourth ventricle; *a* is the posterior pyramid of the left side, forming the outer part of the floor. *b, b'* the vesicular nucleus of the uppermost roots of the spinal-accessory nerve of the same side; immediately below *b'* is a dark oval mass consisting of bundles of longitudinal fibres. The posterior surface of this nucleus with that of its fellow of the opposite side, here forms the point of the calamus scriptorius, and the floor of the fourth ventricle, *f, e, f'*, on

each side of the medial line. *c* is the space surrounding the elongated canal. *d* is the vesicular nucleus or centre of origin of the lingual nerve of the left side; on the opposite side its large and variously-shaped cells are represented. *g g'* are the roots of the lingual nerves. *h* is the raphè.

Now at this level of the medulla oblongata, the surfaces of the spinal-accessory nuclei *f*, *f'*, forming the median floor of the fourth ventricle, were covered with a layer of oil-globules, resulting evidently from degeneration of their superficial portions, which were ragged, uneven, and, as it were, eroded. In the median furrow also their natural substance was completely destroyed, and replaced by a deep column of densely-crowded oil-globules, *e*, which were continuous with those on each side *f*, *f*. The point of the calamus scriptorius was composed entirely of this fatty column; and a similar degeneration, but in a less degree, extended to the corresponding parts a little below, as well as a little above, where it partly affected the nuclei of the pneumogastric nerves.* All the remaining, deeper portions of the spinal-accessory nuclei *b*, *b'*, were scattered at intervals with isolated oil-globules of considerable size; and the space between them, *c*, surrounding the canal, contained them in still greater numbers. The posterior pyramids *a*, *a*, were likewise interspersed with the same kind of globules, which were to be found, also, more sparingly scattered even through the restiform bodies. The cells of the spinal-accessory nucleus were normal, many of them being filled, as usual, with dark-brown, or nearly black pigment granules.† The lingual nuclei contained no oil-globules, and their cells were normal in shape and size, but enclosed rather more pigment than usual.

NEW OBSERVATIONS UPON THE MOVEMENTS OF THE LIVING OR GERMINAL MATTER OF THE TISSUES OF MAN AND THE HIGHER ANIMALS.

By Lionel S. Beale, M.B., F.R.S.

Preliminary Communication.

The movements of the matter which I have distinguished as living or germinal matter of many of the lower ani-

* It must be recollected that the nucleus of the pneumogastric nerve is the upward continuation of the spinal-accessory nucleus from about the *calamus scriptorius*. (See my "Researches on the Medulla Oblongata," Phil Trans. 1858.)

† Without knowing that a number of dark pigmentary cells are always found in the spinal-accessory nucleus in a state of health, these bodies might be mistaken for the products of disease. It is a curious fact that I have not found them in the corresponding nuclei of mammalia. (See my Researches, loc. cit.)

mals and vegetables, are well known. I have endeavoured to show that the power of movement resides in the living particles themselves, and have expressed the opinion that these movements cannot be explained by physics or chemistry. Particles far too minute for us to see by the highest powers to be obtained (5,000 diameters), must possess the inherent moving power, the effects of which we observe in the changes occurring in the form of the masses of living matter which are but the aggregate of such minute living particles. It seems to me probable that the most minute living particle which it is possible to conceive, is a spherule, and that this spherule is capable of altering its form. I believe that the alteration results from the influence of wonderful inherent internal powers, of the nature of which we know nothing as yet, which wonderful powers may at least, for the present, be termed "vital," to distinguish them from physical and chemical properties.

I am now in a position to extend this view to the germinal matter of tissues.

I have already stated, that living matter possesses the power of altering its form, and movements have been observed by Brücke and many others in white blood, chyle, and lymph, corpuscles. They can be seen easily in pus and mucus corpuscles, young epithelial cells,* the nuclei of the corneal tissue as shown by Kühne, and indeed the germinal matter of most of the tissues of the higher animals, as shown by the difference of form exhibited under different circumstances, and the existence of protuberances, buds, or offsets. Every one is familiar with these movements in the lower forms of life, but the general conclusion I have arrived at, that the matter which is the seat of these movements is the living or germinal matter, as contradistinguished from the lifeless formed material, is not yet generally admitted.

I have already described, with some care, the movements occurring in the germinal or living matter of the mucus corpuscle from the human throat, and have stated that I had "seen an entire corpuscle move onwards in one definite direction for a distance equal to its own diameter or more."—(British Medical Journal, March 28th, 1863.) Often, as the mass of germinal matter moves, it leaves a sort of thread behind it. This thread I consider to be the lifeless formed matter which results from the death of a portion of this living, moving, growing mass under, certain conditions.

Now just as this living matter of the mucus corpuscle moves

* See my paper in the British Med. Journ., March 28th, 1863.

away, leaving its formed material behind it, so the white blood corpuscle may move and leave fibrin behind it, and the germinal matter of fibrous tissue may leave, as it moves in a definite direction, its proper formed material, which gradually undergoes condensation into fibrous tissue.*

I have evidence to prove that the formed material of many other tissues is produced in precisely the same way. I shall shortly publish the results of observations which establish this most important fact in fibrous tissues generally, in lymph, in the fibrinous clot, and in the formation of connective tissue, in muscle, in all nerves and nervous centres (see also my paper on "The structure and formation of the so-called apolar, unipolar, and bipolar nerve cells of the Frog," Phil. Trans., 1863†), in the formation of dentine and in many other tissues; so that I feel convinced the fact is a general one, and that I shall have no difficulty in proving it by demonstrating its results in the tissues of any living being.‡

That my meaning may not be misunderstood, I will particularly adduce the case of striped muscle and elastic tissue. Köl-liker maintains that in the formation of the latter tissue nuclei take no direct part. I can affirm that what are called nuclei take the same part in the formation of this as in the case of other tissues. I can very clearly demonstrate the nuclei in the ligamentum nuchæ of the lamb, in specimens which have been immersed in carmine.

The mass of germinal matter of muscle seen in Fig. 1, which occupies the interval between *b* and *c*, has moved there, for

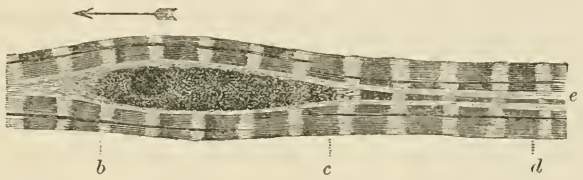
* The two masses resulting from the division of a mass of germinal matter move in opposite directions; and thus in a mass of tissue, when movement can occur only in opposite directions, the oval masses of germinal matter will alternate with each other throughout the texture; one end of the mass is often blunt and rounded, while the other gradually tapers into the thread of formed material.

† This memoir will shortly be published in a separate form.

‡ In many secreting cells, as, for example, in the long mucus secreting cells upon the fauces of many of the reptiles (they are very large in the boa), the so-called nucleus, or mass of germinal matter, is near the lower or attached portion of the cell; while, extending towards the surface, is the long wide tube of the cell, often open at its free end, which contains the product of secretion. In this case the pabulum is taken up from below, and absorbed by the deep surface of the small mass of germinal matter. At the opposite surface the germinal matter undergoes conversion into formed material, which pushes that which was produced before it onwards towards the free extremity of the cell. The germinal matter remains stationary, but movement in a definite direction is given to particles resulting from its death. Ciliary movement is probably a phenomenon very closely allied to the above; and, I believe, in the absorption of matters by the cells of the villi a similar change occurs, but, of course, in the opposite direction. The position of the mass of germinal matter (nucleus), with respect to one or other extremity of the cell, will depend upon the position of the pabulum. The nucleus is usually nearest that extremity of the cell which absorbs the pabulum.

it was once in the space between *c* and *d*. As it moved, part has undergone conversion into the contractile formed material *e*, and a continuation of the contractile material would, if the muscle had lived, soon have occupied the position between *b* and *c*, while the mass of germinal matter would have moved still further on. At the same time that it advances it absorbs nutrient matter, so that much formed material or contractile tissue may be produced without the germinal matter altering in size.

Fig. 1.



Portion of voluntary muscle, from the midst of an elementary fibre of the frog, very highly magnified, showing the manner in which the contractile tissue (fibrillæ) is formed from the germinal matter. Mass of germinal matter occupying the space between *b* and *c*. It has moved to its present position, and once occupied the interval between *c* and *d*. *e*. Contractile tissue, which was recently in the state of germinal matter. Over *c*, the conversion of germinal matter into formed material (contractile tissue) is actually proceeding. The mass of germinal matter is moving in the direction indicated by the arrow.

Fig. 2 is a portion of an elastic fibre of the ligamentum nuchæ, with its mass of germinal matter *a*, which is moving in the direction of the arrow, while part gradually undergoes conversion into the elastic material of the fibre which in this way becomes thickened.

Fig. 2.

Portion of a fibre of yellow elastic tissue from the ligamentum nuchæ of the lamb. The mass of germinal matter *a* is moving in the direction of the arrow. At the posterior part the formation of yellow elastic tissue is proceeding; the fibre, increasing in thickness as new formed material results.

Similar phenomena occur in a clot in which fibrous tissue is to be formed. Masses of the germinal matter, probably descended from the white blood corpuscles, move in segments of circles and form fibres of connective tissue.

The details upon which I rely for establishing this most

important fact, in connection with the formation of the tissues, are most interesting, and will, I think, especially when considered in connection with many phenomena familiar to all observers, be admitted to be very strongly in favour of the general conclusions I have been led to adopt.

ANATOMICAL AND PHYSIOLOGICAL CONTROVERSIES.

It is doubtful if at any previous period so many incompatible views were entertained upon questions of paramount scientific interest and importance, as at the present time. Nor are the differences which exist slight or unimportant, affecting mere matters of detail. Great principles, which can be very clearly enunciated in few words, are involved, and many of these influence the very foundations of physiology. Careful observers are at variance upon matters of observation, and many questions of the utmost importance to physiology and medicine are in the most unsatisfactory state. Rival observers originate, and rival pupils support, opposite doctrines; warm and exciting conflicts disturb the steady quiet necessary for true advance, and continual working in circles, retards real scientific progress. In no branch of science is this very unsatisfactory state of things more apparent, than in minute anatomy, and as a necessary consequence, grave doubts are cast upon the value of direct anatomical observations as a means of discovering truth, and the great differences among workers upon fundamental principles, foster, and, to some extent, justify, scepticism on the part of readers and learners.

A careful perusal of many of the articles in small print in Kölliker's Anatomy, for example, will enable any one to form some notion of the terribly conflicting views now entertained upon what we all look upon as simple anatomical questions affecting the very foundation of physiological science. If the reader will be at the pains of comparing some of these articles in the last with the corresponding ones in the first edition issued of this work, he may, perhaps, be able to form some idea of the character the work will assume ten years hence. For if observers increase in number as they have increased lately, and if, as is really not improbable, there yet remain many p o s s i b l e views upon every subject which have not yet been put forward, it will be necessary to devote an entire volume to the consideration of each separate tissue and organ. We cannot but feel that it is the duty of all who work at minute

anatomy, to make an effort to arrive at definite conclusions on wide points of general interest, and it seems to us, that many of the complex views now held might be simplified without difficulty.

It is a remark that will, perhaps, be received with some surprise, but it is nevertheless true, that conflicting views result principally from insufficient work. Observers often write upon controversial subjects after devoting a very short time to observation, and many instances could be alluded to, in which conclusions, resulting from years of thought and labour, were set aside by authorities after a most imperfect consideration of the question at issue. After a time the true view is, of course, revived, and finally accepted, but what time and labour have been for ever snatched from true progress in the meanwhile !

It is well known that Henle has recently reopened the whole question of the anatomy of the kidney. If his conclusions are correct, the views which have been taught upon the anatomy and physiology of this organ during the last twenty years, will require to be modified greatly, and not only so, but the pathology of renal diseases must be re-studied from a new point of view. Now surely there are specimens already prepared in various collections, which would confirm or refute Henle's observations. Would it not be possible for those interested in this question to send one or two specimens to some general meeting of anatomists, which specimens could be examined by the observers present? Professor Henle would doubtless send some of those upon which his inferences rest. We do not say that the question at issue might in this way be conclusively determined and completely set at rest; but, at any rate, observers would be in a far better position to judge of the correctness of these new observations than they are at present. Professor Henle is doubtless aware that such an arrangement as he supposes to exist in man, in the horse, and in the pig, cannot possibly exist in the newt, in which animal a tube can be traced in every part of its course quite uninterruptedly from the Malpighian body to its terminal portion, and injections can be forced quite through it from the Malpighian body. Surely this question of the existence of tubules without efferent ducts in the particular kidneys above named, is one which might be determined by any observer who was an adept at injection, and would devote to it a few weeks' careful work.

Even greater differences of opinion still exist upon the anatomy of the liver, and many anatomists do not admit the existence of inter-lobular ducts in all the inter-lobular fissures,

and few agree that any ducts pass into the lobules themselves. This is an anatomical question, which perhaps can only be fully elucidated in the course of a controversy between those who have studied the subject and have arrived at different conclusions.

But differences of opinion equally striking exist among living observers with regard to the minute structure of almost every organ and tissue, not only of the higher but of the lowest and simplest organisms. The physiological views deduced from anatomical observation and physiological experiment, are equally conflicting.

The connective tissue theory, the distribution of nerves, and their relation to the anatomical elements of the tissues, especially to connective tissue corpuscles and epithelium, the mode of formation of yellow elastic tissue, the mode of the distribution of nutrient pabulum to connective tissues, the manner in which nutrient matter is absorbed by the columnar cells of the villi and passes into the lacteals, are instances of great anatomical questions upon which the most conflicting and incompatible views are entertained by different observers, and taught in different schools.

Yet, with all this doubt upon elementary matters, and in spite of the difficulty of arriving at the truth upon mere questions of fact, it is astonishing, with what outrageous confidence opinions are expressed and assertions made, upon matters which real students would truly regard as the highest and most difficult problems to be solved. We have a whole army of conscientious observers utterly puzzled as to what happens when the cell of a leaf or the germ of a simple vegetable organism increases in size by growth; while, on the other hand, the most abstruse and difficult questions which ever have been, or which ever can be, presented to the mind of man to solve, are disposed of by another class of philosophers in the most off-hand manner possible, and rendered clear to the comprehension of a child, as if by magic. There is no longer any necessity for naturalists to spend their lives in studying the life-history of a few species, or for minute anatomists and physiologists to peer into the inmost recesses of living creatures. Living organisms upon this earth exhibit in the changes which take place in them nothing different to what occurs in stones, in planets, and in suns. We live in an age which to posterity will appear the most marvellous the world has ever seen. The views now put forward by most popular philosophers are destined to stand for ever unshaken. The end of all knowledge has been

reached. A new philosophy has appeared, as perfect as that which preceded it was incomplete. Our scientific successors will doubtless have reason to respect our work far more than we have to respect that of a former generation. We know how much that was believed by our predecessors has been demonstrated by us to be unsound, but those who succeed us will doubtless find that our views will stand unshaken in their beautiful and simple perfection. Those now living have seen vast problems explained, so that those who follow will have but to admire, and contemplate, and wonder at what has been done by philosophers of the present era.

There appeared to some naturalists and physiologists difficulty in explaining how Darwinian monads first became living, and although it is obvious that a few particles of organized matter coming together might produce a living creature, it was somewhat difficult to account for the formation of the organized particles before life began upon the earth. But a more sublime discovery, and a more stupendous generalization, has been made by the physicist, and henceforth biology and physiology will be obsolete; for biological phenomena henceforth belong to physics. By life we merely understand certain changes, physical, chemical, electrical, thermal, &c., which can be precisely imitated in the laboratory with precisely similar results. Vital power no longer excites the speculations of the physiologist, or the wonder of the profound metaphysician. Every tyro now knows that life is but another form of motion, and that the changes taking place in living things differ from those occurring in stones only in degree. The same mighty forces are at work in the organic and in the inorganic world. The formative power of the living organism is but a form of "heat," without which nothing living could exist. We know the conditions under which the crystal forms, and we know the circumstances under which the egg will develope.

Particles of inorganic matter arrange themselves in a certain order, and give rise to certain definite and constant forms. Nuclei arrange themselves in rows, become invested with surrounding substance, and thus a living tissue is built up! The complex tissues of the higher animals scarcely differ more from the simplest organisms than the latter from inorganic matter. In all things changes occur. In living beings these changes take place with greater rapidity, owing to the different conditions under which the matter is placed.

The real nature of the mysterious "vital forces" of a former day is now fully explained by the new discoveries of the physicist, and the true relation of these forces to the one great primary agent, simple motion, has been incontrovertibly established. In fact,

no one who is able to appreciate the marvellous results of recent physical investigation can fail to accept the inevitable conclusion, that life itself is but another form of that marvellous energy we receive from the sun. Vital action is only motion peculiarly conditioned. Like other kinds of force, it is neither created anew nor can it be annihilated. When a living thing dies, its equivalent of constructive organizing force changes its form, and either returns again to the more simple state of heat, or manifests itself in the vital actions of some lower forms of life. The living fungus growing upon the dead branch contains its equivalent of that energy which was working in the branch when it was alive. Thus it is that there is an ever-changing expression, but a never-varying quantity of force. It may be fungus life, tree life, animal life, or man life, but equal weights of fungus, of tree, of animal, and of man, will contain of course different equivalents of force.

All this energy, which is being continually supplied to our earth, and is here conditioned in so many different ways, emanates from the sun. He not only moves the steam machine made by man, but he forms and then moves the living machine. He is the chemist, who not only works in our cell laboratories, but forms them. At his bidding the cell laboratories multiply in number, and he gives to each a portion of his mighty energy. He dissolves, he evaporates, he distils, he condenses, he dries, he ignites. He arranges the retort, he applies the heat, he rises in the vapour, he falls in the distillate. By his influence, elements which were firmly linked in combination silently separate, take up their appointed positions, and assume relations which they never had known before. Some of his rays disappearing in the process, and assuming the condition of latent internal energy, new compounds result, which the synthetical powers of the most expert modern chemist have scarcely yet enabled him to produce in perfect purity; but without doubt defects in the existing chemical processes will soon be remedied, and protein compounds, now regarded as peculiar to the living organism, will be obtained in a state of purity in a crystalline form.

Nor does the sun merely play the part of cell laboratory builder, cell laboratory multiplier, and cell chemist. His work is more general and far more varied than this He *forms* the most simple and most complex structures of living beings. The petals of the rose as well as its delightful perfume, the shell of every diatom, the spicules of a hundred sponges, are his workmanship. He oscillates in the monad, he vibrates in cilia, he revolves in the rotifer. The human brain, like the forest, is

built by him, and we ourselves consist of nothing more than inorganic matter and a fraction of his energy. The formation of tissue, the action of all the different organs, their disintegration, the different phenomena observed in the same structures in health and disease, are easily accounted for by the different conditioning which the sun's force undergoes as it passes through our bodies. The soul-inspiring strains of the poet and the wisdom of the sage, the ravings of the madman, are but flickerings of rays emitted by the sun. It is very easy to understand how a minute particle of matter, so very light that many hundred times its weight would fail to turn the most delicate balance ever made, and so very minute that it is scarcely visible under any magnifying power, being supplied with a fraction of such marvellous energy, must form into a plant, or a tree, or a man. But we need not stop here, for we know how asteroids, moving with intense velocity, meet together and form suns. The immense energy which now takes the form of heat instead of motion, radiating into space and falling upon distant worlds, is conditioned in its halting-places, and becomes in its turn vegetables and animals, which afterwards melt into air, and thus give up again every fraction of force they had received.*

It is needless to pursue further the results of modern discoveries upon our notions of life. The term 'vital power' can no longer be used, for as it emanates from, and is produced by, the sun, it will be henceforth better known as sun-force. All things living from the monad to man are children of the sun. We have thought it well to give an outline of these views which have been recently rendered so clear, because the nature of vital actions has been so fully explained that it is not likely to become again a subject of controversy.

But there still remain many physiological questions of the utmost practical importance to those who have to study and treat disease, which are, as yet, undecided; and there can be no doubt that some of these might be in great measure cleared up by fair but temperate discussion. There are many simple remedies, with regard to the action of which upon the organism much yet remains to be learned. We are not yet acquainted with the action of the mineral acids and alkalis, and we all know that the very same cases would often be treated with acids by some physicians, and with alkalies by others. Then there is the all-important question of the external and internal action of alcohol in disease. What differences of opinion there are

* See Professor Tyndall's Lectures at the Royal Institution, on "Heat considered as a Mode of Motion," pp. 433, 434.

upon the action and practical value of this remedy. It ought to be possible to obtain some principle which would guide us as to the quantity that should be given in various conditions. Many practitioners have asserted that no patient, under any circumstances, should take in twenty-four hours more than a certain number of ounces. Some may think that those who enforce such dogmas perhaps rely too much upon mere opinion, since it is obvious that the weight of the individual, the activity of the processes of respiration and secretion, must affect the proportion that ought to be given. It is, therefore, clear, that the quantity which should be given must vary in different cases. Here is a practical question of the utmost importance, which can only be settled by discussion.

Temperate discussion between those who really work and earnestly think upon these great questions, will alone enable us to arrive at the truth within a reasonable period of time. Those who put forward conclusions usually support them by facts and arguments, but the real value of these facts and arguments can only be ascertained by the most careful and stringent examination of the evidence upon which they rest. In many of the questions alluded to, it is clear that at the present day two opposite opinions may, so to say, be tenable; that is, the facts in favour of each seem to be of nearly equal value; but it is clear that directly opposite conclusions upon a given question cannot both be true, and therefore new work is required. There is no method by which the direction in which new work should proceed is so soon elicited or so clearly defined as by controversy. In this way alone can opinions which are true be fully substantiated and proved to be true, and worthy of general reception. In this way alone can conclusions which are erroneous, be disproved, and demonstrated to be unworthy of confidence. By free discussion alone can hopeless working in a circle be stopped. It is, therefore, the intention of those connected with this journal to insert from time to time controversial papers of scientific or practical interest. The differences entertained will be expressed as clearly as possible, the papers will be freely illustrated, and every effort will be made to bring the points under discussion to a distinct issue.

AN ANATOMICAL CONTROVERSY.

THE DISTRIBUTION OF NERVES TO VOLUNTARY MUSCLE. DO NERVES TERMINATE IN FREE ENDS, OR DO THEY INVARIABLY FORM CIRCUITS AND NEVER END?*

By Lionel S. Beale.

By no means the least interesting of recent anatomical controversies is one which relates to the distribution of nerves to the voluntary muscle of vertebrate animals. In this particular controversy I take the warmest interest, for the subject was reopened by me in 1859 (see paper in Phil. Trans. 1860, p. 611). The conclusions I then arrived at were opposed to the views entertained by most authorities, especially Kölliker, Gerlach, and Kühne. My paper was followed by a communication from Kühne, in which he supported, by investigations upon the breast muscle of the frog, conclusions advanced by him previous to the publication of my paper, in favour of the view, that the nerves in insect muscle terminated in ends beneath the sarcolemma—in fact, were continuous with rows of nuclei which lie amongst the contractile tissue. In a note to my first paper (Phil. Trans. 1860, p. 611), I have stated that in the muscles of the blow-fly "the nerves do not penetrate into the interior of the muscle, but are connected with some very large spindle-shaped cells on its surface. These are very difficult to demonstrate, but I have been able to preserve some specimens which show this point very clearly." A month after Kühne's paper appeared, a memoir was put forward by Kölliker, in which he agreed with Kühne as to the termination of the nerves by ends, but with me as to the fine nerve fibres being upon or external to the sarcolemma, instead of penetrating through this membrane and coming in contact with the contractile tissue. Here then are three inferences upon this question of anatomical observation utterly incompatible with each other. It might be said that all are wrong; but it is an absolute necessity that two are wrong, since no two of the following arrangements can coexist:—

1. That the nerves terminate in ends external to the sarcolemma.
2. That the nerves terminate in ends beneath the sarcolemma.
3. That the nerves do not terminate in ends at all.

* The reader is recommended to study Plate XI, with the explanation, before reading the paper.

Within the last year many new memoirs have appeared, in which different, and in some cases new views are put forward; but it is undoubted that the authors all differ from me in a greater degree than they differ from one another.*

Such opposition upon a question of fact is greatly to be regretted, and it seems to me that the only way in which the question can be cleared up is by renewed investigations and increased work.

I for my part cannot yield, for the simple reason that I have seen what I have figured, and have indeed found no difficulty in following fine fibres structurally continuous with dark-bordered fibres for a long distance beyond the point where Kühne, Kölliker, and others, make them end. Moreover, my specimens show the arteries and capillaries as well as the nerves, and I have now worked at this anatomical point so long and so hard, that I have a right to ask that my opponents should prepare the specimens by the same process that I have followed, and give drawings of what they observe. I would then reply by giving drawings of the very same structure, showing what I have seen. Independent observers would then be in a position to judge between my opponents and myself.

The great question is as to the existence or absence of ends

* Kühne, Ueber die peripherischen Endorgane der motorischen Nerven.—Leipzig, 1860.

Theodor Margó, Ueber die Endigung der Nerven in der quergestreiften Muskelsubstanz.—Pest, 1862.

Kölliker, Untersuchungen über die letzten Endigungen der Nerven.—1862.

Naunyn, Ueber die angeblichen peripherischen Endorgane der motorischen Nervenfaser, in Reichert und Du Bois Reymond's Archiv. 1862, p. 481.

L. Beale, Further observations on the distribution of nerves to the elementary fibres of striped muscle.—Phil. Trans., June, 1862.

Krause, Ueber die Endigung der Muskelnerven.—Henle and Pfeufer's Zeitschrift, 1863, p. 136.

Th. W. Engelmann, Ueber die Endigungen der motorischen Nerven in den quergestreiften Muskeln der Wirbelthiere.—Centralblatt f. d. Medic. Wissensch, 1863.

L. Beale, "On the anatomy of nerve fibres and cells, and on the ultimate distribution of nerve fibres."—Quarterly Journ. of Mic. Science, April, 1863.

L. Beale, Further observations in favour of the view that nerve fibres never end in voluntary muscles.—Proceedings of the Royal Society, June 5th, 1863.

L. Beale, Remarks on the recent observations of Kühne and Kölliker upon the termination of the nerves in voluntary muscle.—Archives of Medicine, vol. iii., p. 257.

Th. Wilhelm Engelmann, Untersuchungen über den Zusammenhang von Nerve und Muskelfaser.—Leipzig, 1863.

Kühne, Ueber die Endigung der Nerven in den Muskeln.—Virchow's Archiv., Band 27.

Kühne, Die Muskelspindeln.—Virchow's Archiv. Band 28.

Kühne, Der Zusammenhang von Nerv. und Muskelfaser.—Virchow's Archiv., Band 29.

or terminal extremities. My original paper was to show that no ends were demonstrable—that from every oval "nucleus" a fibre could be traced in opposite directions, and I was compelled to infer that the fibre was endless. The appearance was that of a network. Some appear to have concluded that by the term "network" I meant that amongst the muscular fibres existed a common network, with which, at various points, dark-bordered fibres were continuous, just as small arteries and veins are connected with the capillary network at various points. This is an erroneous inference, as will be seen by reference to my first drawings. The network is like the network of lace, every thread being composed of finer threads, but in no case does a single thread pass completely round one mesh or interval. It may form part of the boundaries of many spaces, but not the entire boundary of a single space. Subsequent research in different tissues, and in many different animals, has convinced me, not only of the truth of my original conclusions respecting the distribution of nerves to muscle, but of what is far more important; and I will now take up the question in a much broader way. I assert that nerves do not terminate anywhere in free ends; and I am ready to study by my own processes of investigation any tissue of any common animal of any class that a fellow-worker may select, and discuss this large question by the publication of drawings; or if several observers will meet and agree to investigate any one particular tissue, and publish their drawings and results by a certain day, of course allowing sufficient time for investigation, my drawings and conclusions shall be in print at the appointed time. In this way such disputed points would soon be brought to a distinct issue.*

It is impossible to over-estimate the importance of this general question, for in it is involved the typical arrangement of every nervous instrument. So far from there being distinct ends I believe that in all cases "complete circuits" exist, and that in these circuits are included central nerve cells and peripheral nerve cells, generally termed nuclei, which are connected by intervening fibres. The course of any given fibre may be extremely complicated, and there may be many minor circuits connected with the greater one, but in all cases there is a circuit. A nerve never ends.

In the controversy connected with the arrangement of nerves in voluntary muscle, I think I have been somewhat incon-

* It is of course absolutely necessary that some species, easily procurable in all parts of Europe, should be chosen, and that drawings be published. The illustrations can be executed in any way the observer may desire.

siderately dealt with by some of my friends in Germany. Kühne especially has unceremoniously disposed of me by saying that the nerve fibres I described in 1860 are "vascular nerves." Now if any one will refer to my drawings (Phil. Trans., 1860), he will see the vessels as well as the nerves delineated, and if he will count the nerves and vessels, and observe the position of the nerves and vessels, and the vascular nerve distributed to a capillary in one of the figures, he will indeed be puzzled at the conclusions of my friend.*

Again, Kühne considers that I have mistaken connective tissue and its nuclei for nerve fibres and their nuclei. He sets me right by telling me that a fibre which I have seen exhibiting a very definite structure and arrangement, is nothing but indefinite connective tissue. Now I have very fully considered, in several of my papers, the relation of nerves to connective tissue, and the distinction between the finest nerve fibres and connective tissue, so that I need not minutely discuss this part of the question here. Let the reader refer to my drawings (1860), and he must conclude, either that the fibres I have delineated are not connective tissue, or that I have most wilfully misrepresented connective tissue.

Again I would venture to remark, that not one of my opponents has condescended to investigate the structure in the manner I stated was necessary to see the appearances I delineated, namely, by soaking the tissue in syrup or glycerine, so that not one of those who correct me has seen the fibres I have described. To this day the finest nerve fibres I demonstrated in the diaphragm of the mouse, have not been seen by those who have since taken up the same investigation. And why, I would ask, do not my opponents study the same muscle in the white mouse. I have tried many others, and have found that the diaphragm is most easy of investigation. If this muscle be carefully studied, such plexuses of dark-bordered and fine pale nerve fibres will be found as will astonish the observer.

I have very frequently dwelt upon the importance of injecting the capillary vessels with transparent injection; and before I commenced my first paper I had thoroughly convinced myself,

* Strange to say that at the time my friend passed these severe criticisms he had not seen my drawings in the Phil. Trans. for 1860, which however were not published until 1861—Kühne's memoir is dated 1862. In page 28, after referring to the abstract of my paper published in the Proceedings of the Royal Society, he says: "Deren Original mir leider, nicht zugänglich ist." As it is evident my original drawings in the Phil. Trans. are not known to many of those who have been writing papers upon this anatomical question in Germany, I propose shortly to republish the most important of them.

Plate VI.

To illustrate Mr. Lockhart Clarke's Observations on the Structure of the Cord in a case of Wasting Palsy, and Dr. Beale's Observations on Mucous Casts from the Uriniferous Tubes, pages 39, 40, 56.

Fig. 27.—A similar section opposite the middle roots of the second dorsal nerves. The morbid area, *b, b,* after having diminished gradually, and nearly disappeared, again increases in extent. At *b,* the posterior commissure, and parts surrounding the canal, were quite broken up, consisting of bits of nerve fibres, of granules, and of numerous corpora amylacea. (See § 9.)

Fig. 28.—A similar section at the upper roots of the second dorsal nerves. The transparent patches of degeneration are prolonged backward on the right side, as well as on the left; and for a few lines upward along the cord, the whole inner portions of the cervix cornu were destroyed by irregular patches of granular degeneration, *b, b′, b″, b‴,* like a piece of moth-eaten cloth. At *s,* an enlarged and unhealthy-looking blood-vessel is seen entering through the anterior median fissure, and crossing the lower area of degeneration. (See § 9.) This sketch has been shaded to represent the grey substance, and show the morbid spaces more distinctly. (See § 9.)

Fig. 29.—A similar section opposite the lower roots of the first dorsal nerves. The morbid area, *b′, b″,* acquires its largest dimensions. (See § 10.)

Fig. 30.—Large nerve-cells, variously affected by atrophy and degeneration. (See § 11.)

Fig. 31.—Casts composed of transparent mucus from the straight portion of the uriniferous tubes from a case of renal irritation. Magnified 150. (See page 56.)

Plate VII.

To illustrate Dr. Marston's Paper upon Aneurism of the Aorta, communicating with the Pulmonary Artery, page 63.

Fig. 1.—Loud bronchial and crepitant râles heard over these parts. 2. Region of hepatic dulness. 3. Dull from the presence of fluid (Ascites). 4. Resonant. 5. Centre of cardiac dulness, and seat of purring tremor. 6. Apex beat to the left of nipple; seat of a double murmur (the diastolic one

being faint). 7. Double murmurs of maximum intensity (the diastolic being the loudest) heard in the direction of the arrows. 8. Seat of an undulating impulse of the right heart.

In the area marked out by the figures 6, 7, 8, cardiac murmurs were audible.

Fig. 2.—6 marks the situation and extent of splenic disease.

Plate VIII.

To illustrate Dr. Ogle's Paper on Laceration of the Lower part of the Right Internal Jugular Vein, caused by Fractured Portions of Clavicle, page 125.

The lacerated opening in the internal jugular vein, is seen at *a*.

Plate IX.

To illustrate Dr. Beale's Paper upon the Branching of Nerve Trunks, and the Sub-division of the Individual Fibres composing them, page 127.

Fig. 1.—The course of the branches in a large compound nerve-trunk, or of the fine fibres in a very fine nerve-trunk, at the point where a branch is given off at right angles.

Fig. 2.—The course and division of individual dark bordered nerve-fibres, showing that one of the fibres, resulting from the sub-division of a fibre, passes on in the direction of the original fibre, while the other passes into the branch which diverges from the trunk. *a* and *d* may be commissural fibres between two sets of cells at the periphery, or between two sets of central cells. The arrows indicate the direction which the nerve-currents in the different fibres may take.

Fig. 3.—Branching of a fine nerve-trunk, from the pectoral muscle of the frog, showing the course of the respective dark-bordered fibres in the trunks, and their division.

Fig. 4.—A fine trunk composed of very fine nerve-fibres from the mylo-hyoid muscle of the hyla viridis, magnified 1,800 diameters. The general arrangement in the case of these very fine fibres, is the same as that observed in dark-bordered nerve-fibres, and in compound nerve trunks.

Fig. 5.—Diagram to illustrate the manner in which the nerve-fibres distributed to the papillæ of the tongue of the frog are arranged. It will be observed that the papillæ are con-

nected to one another by commissural fibres, while some of the fibres of the nerve-trunk are not connected with all the papillæ figured. There are also fibres in the trunk which are not distributed to any one of the three papillæ in the figure. The nerves end in a net-work or plexus, at the summit of each papillæ.

Fig. 6.—A nerve dividing into three branches. From the breast muscle of the frog. At *a* and *b*, individual dark bordered fibres are seen to divide, the fibres resulting from the sub-division passing onwards in different branches. See fig. 2.

Fig. 7.—Fine trunks resulting from the sub-division of a small branch of the nerve distributed to the breast muscle of the frog.

Plate X.

To illustrate Mr. Lockhart Clarke's Pathological Observations in a Case of Epilepsy, accompanied by Diabetes, page 146.

Fig. 1.—Substances found in a rust-coloured mass about the size of a hazel-nut, near the middle of the inner third of the right optic thalamus.

a. Oblique rhombic prism of hematoidin. *b*. Three crystals of the same embedded in mass of fat globules. *c, d, e, f*. Amorphous granules of hematoidin interspersed through oil globules of different sizes. *b, f, g', h*. Oil globules, stained of a yellow or brown colour, by a solution of hematoidin. *j, l*. Exudation corpuscles, consisting of minute globules of fatty matter. *k*. Acicular and radiating crystals of margarine.

Fig 2.—A transverse section of the medulla oblongata on each side of the median line, a little above the point of the calamus scriptorius.

f, e, f'. The median portion of the floor of the fourth ventricle.

a. The posterior pyramid of the left side, forming the outer part of the floor.

b, b'. The vesicular nucleus of the uppermost roots of the spinal-accessory nerve of the same side; immediately below *b'* is a dark oval mass consisting of bundles of longitudinal fibres. The posterior surface of this nucleus with that of its fellow of the opposite side, here forms the point of the calamus scriptorius, and the floor of the fourth ventricle, *f, e, f'*, on each side of the medial line.

c. The space surrounding the elongated canal.

d. The vesicular nucleus or centre of origin of the lingual

nerve of the left side; on the opposite side its large and variously shaped cells are represented.

g, *g'*. The roots of the lingual nerves.

h. The raphè.

At this level of the medulla oblongata, the surfaces of the spinal-accessory nuclei *f*, *f'*, forming the median floor of the fourth ventricle, were covered with a layer of oil-globules, resulting evidently from degeneration of their superficial portions, which were ragged, uneven, and, as it were, eroded. In the median furrow also their natural substance was completely destroyed, and replaced by a deep column of densely-crowded oil-globules, *e*, which were continuous with those on each side *f*, *f*. The point of the calamus scriptorius was composed entirely of this fatty column; and a similar degeneration, but in a less degree, extended to the corresponding parts a little below, as well as a little above, where it partly affected the nuclei of the pneumogastric nerves. All the remaining, deeper portions of the spinal-accessory nuclei *b*, *b'*, were scattered at intervals with isolated oil-globules of considerable size; and the space between them, *c*, surrounding the canal, contained them in still greater numbers. The posterior pyramids *a*, *a*, were likewise interspersed with the same kind of globules, which were to be found, also, more sparingly scattered even through the restiform bodies. The cells of the spinal-accessory nucleus were normal, many of them being filled, as usual, with dark brown, or nearly black pigment granules. The lingual nuclei contained no oil-globules, and their cells were normal in shape and size, but enclosed rather more pigment than usual.

Plate XI.

To illustrate Dr. Beale's Observations upon the Anatomical Controversy concerning the distribution of Nerves to Voluntary Muscles. Page 160.

Fig. 1.—Pale fibres with peculiar bodies (Endknospen), said to exist beneath the sarcolemma by Kühne. This is a part of Kühne's fig. 7, Plate II., accompanying his memoir "Uber die peripherischen Endorgane der motorischen Nerven." The white substance is said to cease at the sarcolemma, while the axis cylinder is continued beneath as an intramuscular pale fibre with nervenendknospen.

Fig. 2.—A part of one of Kölliker's figures, copied from the Croonian Lecture (May, 1862), published in the Proceedings of

the Royal Society, vol. xii., p. 60. "Termination of a dark-bordered tubular nerve-fibre on a muscular fibre *f*, of the cutaneous muscle of the frog, seen with Hartnach's objective à immersion No. 10, and eye-piece No. 1. *a*, sheath of the nerve fibre continued upon the pale terminal fibres; *c c*, nuclei of the pale fibres; *d*, a nucleus belonging to the muscular fibre *f*, *e e e*, ends of the pale fibres; *b*, nucleus of the dark-bordered fibres. The figure shows the continuation of the dark-bordered fibres into the pale filaments, but fails to exhibit the faint outline and characteristic aspect of the latter."

The fine fibres which Kühne maintains lie beneath the sarcolemma and are intra-muscular, are believed by Kölliker and myself to lie upon the external surface of this membrane, and to be inter-muscular.

Fig. 3.—Division and subdivision of fine dark-bordered fibres on the surface of muscular fibre, showing numerous fine fibres arranged in the form of a network, and nuclei connected with these fine fibres. Two or three of the delicate compound nerve fibres may be traced over the edge of the muscular fibre. Pectoral muscle of the frog, 700.

This figure was copied by myself from a muscular fibre from the very same muscle of the frog (pectoral) as that from which the figures of Kühne and Kölliker were obtained. I have copied exactly what I saw. That the fine and pale, as well as the coarse fibres, are inter-muscular and lie upon the external surface of the sarcolemma, is quite certain, as they may be traced over the edge of an elementary fibre, and may be followed from one to the surface of another.

The fibres considered by Kühne and Kölliker to be single pale fibres really consist of several very delicate fibres, and the appearance which has been regarded as the outline of the sheath, is in many cases caused by the presence of very fine fibres, which mingle with the fine fibres which form the direct continuation of the dark-bordered fibres. By comparing these three figures with each other, it will be observed that the constitution of the "pale terminal fibres" is represented very differently. Much of what Kölliker has figured really seems to constitute the sheath, and the lines which, according to him, represent the outline of the sheath, are, according to me, fine nerve fibres. He and Kühne consider that the fibres really end where they are made to cease abruptly in their drawings. On the contrary, what seem to be ends in my drawings merely represent the point beyond which I could not follow the fibre in the particular specimen figured, in consequence of its ex-

treme transparency. I am confident the fine fibres do not end at all, but that these very fine compound but scarcely visible nerve-fibres pass to other trunks, and thus a network is formed with lax meshes. This network is much obscured by the intermuscular connective tissue, but it lies upon the elementary muscular fibres, just external to the sarcolemma. This network of nerve fibres has not yet been observed in this or other tissues by Continental observers, and is still included in the "connective tissue."

Figs. 4, 5, 6, and 7, are copied from Kühne's paper in Virchow's Archiv., vol. 27, Plate XI. In this paper the intramuscular pale fibres for the most part lose their definite character, and their peculiar nerve 'knospen'—represented in the first paper. The axis cylinder is now lost in a collection of granular matter and nuclei, arranged in the most irregular manner.

Fig. 4.—From the psoas of a rabbit. *a*. Nucleus of the nerve sheath. *N*. Nerve. *H*. Nerve prominence (Nervenhügel). *b*. Nuclei in the nerve eminence. *c*. Muscle nuclei. *d*. Glittering globules in the granular contents of the nerve eminence.

Fig. 5.—From one of the muscles of the eye of a dog. The explanation of the letters as above, *a'*, a nucleus with granular contents × 450.

Fig. 6.—From the gastrocnemius of the rabbit. Explanation as above × 450.

Fig. 7.—Also from the gastrocnemius of the rabbit. The nerve eminence somewhat pushed on one side by the pressure of the covering glass × 450. The explanation as in fig. 5.

[I am of opinion that the nerves and nerve nuclei all lie external to the sarcolemma, not beneath it as represented in these drawings. By Kühne's mode of preparation, the nuclei have been completely dislocated from their original orderly mode of arrangement, and the nerve fibres passing between them have been destroyed. The nuclei of the nerve sheath, marked *a*, are, I maintain, in many instances, the nuclei of fine nerve fibres running in the same sheath with the dark-bordered fibres.]

Fig. 8 is copied from a part of another drawing of Kühne's, published in Virchow's Archiv., vol. 28. It represents the nerves in what Kölliker has termed 'nodular swellings,' or nerve tufts, in the breast muscle of the frog. These bodies contain very young muscular fibres, which gradually increase in size, and thus the width of the muscle is from time to time increased. In this figure the dark-bordered fibre is represented as ter-

minating abruptly in a granular nucleated mass. Neither pale fibres, nervenendknospen, nor nervenhügel, are here represented. They may not be developed yet. But the huge dark-bordered fibre is made to end apparently by a wide mouth, which opens into a space beneath the sarcolemma. The terminal nerve fibre is nearly as wide as the tube of the sarcolemma itself, at the point where it becomes intramuscular!

[The further narrowing of these dark-bordered fibres, and their division into finer branches, which run for a long distance upon the surface of the sarcolemma, beyond the point where Kühne makes them enter, cannot be seen in specimens prepared according to the plan followed by this observer. It is quite certain that dark-bordered fibres do not terminate in any tissue in the manner suggested by this figure. I have seen a complete leash of very fine nerve fibres moving over fine muscular fibres like those represented in this figure.]

Fig. 9.—Portion of an elementary muscular fibre from one of the abdominal muscles of the white mouse, with four dark-bordered fibres (*a*) crossing over its surface; a capillary vessel (*b*) is also seen with fine nerve fibres distributed to it. A few of the transverse markings of the muscle are represented at the left-hand corner of the fibre. Two of the dark-bordered nerve fibres (*c*) pass over the elementary fibre, to be distributed to adjacent fibres. One dark-bordered fibre (over *d*) is lost among several pale nucleated fibres. Pale nucleated fibres are seen ramifying in several places upon the surface of the elementary fibre, but beneath the dark-bordered nerve fibres and capillaries. It will be observed that a fine fibre, which seems at the upper part of the figure to be but one of the outlines of the tubular membrane of one of the dark-bordered fibres, leaves the dark-bordered fibre and passes to the capillary vessel (over *e*). I have so often observed a dark-bordered nerve fibre distributed to muscle, divide into two branches, one of which passed to a vessel, while the other ramified upon the muscle, that I believe the arrangement to be usual, and probably essential.

The capillaries, dark-bordered nerve fibres, and pale nucleated fibres, represented in this drawing, as well as the intervening connective tissue, can be completely stripped off from the surface of the sarcolemma, without tearing that membrane. Of the structures, therefore, represented in this drawing, not one can lie beneath the sarcolemma.

Fig. 10.—A small portion of fig. 9, over letter *f*, much more highly magnified. A portion of the capillary vessel is

marked *a*. One of the dark-bordered nerve fibres *b* is seen over *c* to lose itself amongst the number of pale nucleated fibres. The numerous nuclei in this small space are of the nature of those represented by Kühne as lying beneath the sarcolemma, but as this observer has not injected the capillaries with preservative fluid immediately after the death of the animal, the delicate structure here represented has undergone disintegration, and therefore could not be seen by him.

Figs. 9 and 10 I offer as an explanation of the appearances represented by Kühne in figs. 4, 5, 6, 7, and I think that the nuclei irregularly arranged amongst granular matter and placed beneath the sarcolemma in his drawings, are the nuclei connected with the pale fibres outside the sarcolemma in my fig. 10. It is clear that we are at issue upon a very broad question of fact. The very same nuclei which, according to the observations of Kölliker and myself, lie outside the sarcolemma, are placed by Kühne beneath this membrane.

Plate XII.

Illustrating Dr. Beale's Observations upon the Anatomical Controversy relative to the distribution of Nerves to Voluntary Muscles. Page 160.

Distribution of finest nucleated nerve-fibres to the very narrow elementary muscular fibres of the mylo-hyoid of the little green tree-frog (Hyla arborea), magnified 1800 diameters.

The elementary muscular fibres are marked *g*, *h*, *i*, *k*. *k* is a very young one, slightly stretched; *i* is a fully formed muscular fibre; *h*, another stretched in its central part. The nuclei of these fibres exhibit some differences in size and form. Nucleoli are distinct in all, and in the fibre marked *g* the nuclei, which were coloured by carmine, exhibit three different intensities of colour—the dark central spot, "nucleolus," being most intensely coloured, as indicated by the shading in the drawing.

a is a nerve fibre which was followed over more than twenty elementary muscular fibres from a dark-bordered fibre. One of the subdivisions of this fibre is seen at *f*, where it again runs with a very fine dark-bordered fibre (*o*). The dark-bordered fibre (*o*) was some distance higher up in the specimen, but its place has been altered in order to avoid the necessity for a still larger drawing. Above *b* a nucleus of a very fine nerve fibre is seen. Such nuclei lie upon the surface of the muscular fibres, external to the sarcolemma. The nucleus often appears as

× 215.

× 18.0.

Fig. 5.

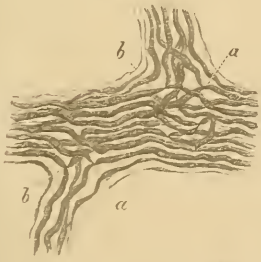


Fig. 7.

Fig. 1.

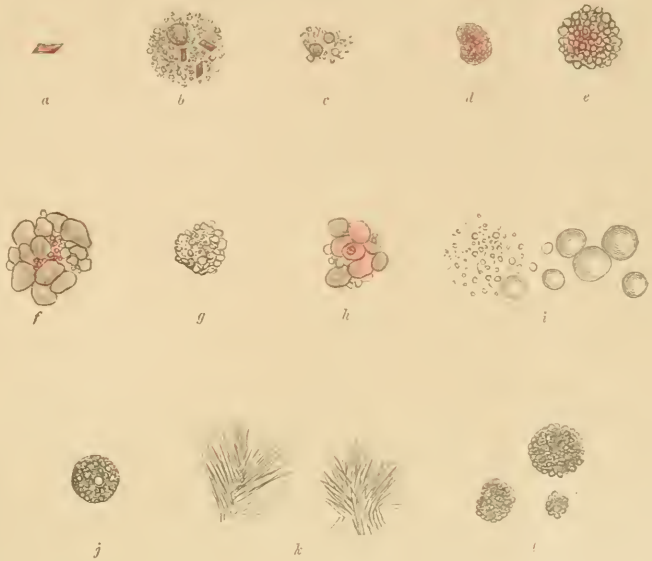


Fig. 2.

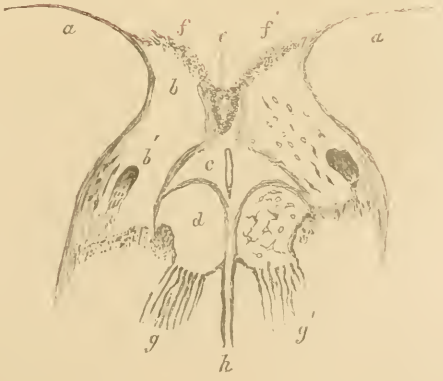
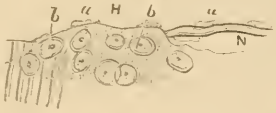


Fig. 5.

× [illegible]

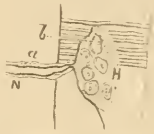
Kühne.

Fig. 6.



Kühne. × [illegible]

Fig. 7.

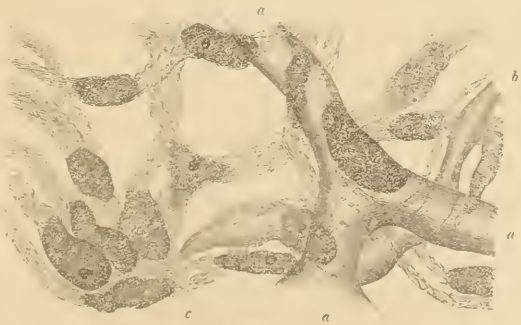


Kühne. × [illegible]

Fig. 8.

Kühne. × 700.

Fig. 9.



Kühne. × 750.

Distribution of [illegible] in the Myo-neural Muscle of the [illegible]. Drawn on the stage by the Author, from a specimen mounted in [illegible] made by Messrs. Powell & Lealand.

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